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A MODERN TREATMENT OF SQUINT.

By J. MYLES BICKERTON, F.R.C.S.,

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IN order to cure a squint it is advisable to treat the cause of the squint. A deviation of the axes of the eyes may obviously be due to a weakness or paralysis of any of the muscles controlling eye movement. Again, congenital absence of the fusion sense may be responsible, or one eye may not see sufficiently well to be able to fix the object regarded.

In a large proportion of squints there is an abnormality in the coordination between accommodation and convergence. Heterophoria is a muscle imbalance which is controlled by the fusion sense. Heterotropia is uncontrolled heterophoria, and diplopia is avoided by suppression of the vision of one eye, a psychological process. Persons who have had binocular vision for some years and develop a squint afterwards, suffer from diplopia.

Ninety-two *per centum* of squinting children suffer from convergent concomitant squint, the eyes swivelling together. In paralytic squint (non-concomitant), of course, this is not so. Now it is to these 92% that I shall devote my main attention in this article.

When the surgeon sets out to cure a squint, his most important duty is to place the axes of the two eyes as nearly parallel as possible (the primary position) and, this having been done, the patient will be most grateful as a rule and will not ask for anything further. It is not a very difficult feat to obtain this happy result, but it is not so easy to keep the eyes in the correct position afterwards, and thus it has been found that unless single binocular vision has been obtained the eyes will often not remain in the primary position.

What is it that maintains the eyes in the primary position?

1. The power to fix an object with each eye simultaneously, that is, Grade I binocular vision,

obtained normally at two to five weeks old, or simultaneous perception.

2. The fusion sense, which develops from one month to eight years usually—Grade II binocular vision: (*a*) plane fusion. The amplitude of fusion may be measured in prism (diopters).

3. Stereoscopic vision, Grade III binocular vision: A psychological cerebral process which blends the two often greatly different images into a stereoscopic single whole (stereos solid). The right eye sees much more of the right side of an object, and the left much more of the left side of an object.

All this shows that the surgeon should be able quickly to analyse the factors underlying any squint, and this requires a careful and systematic routine.

Squints will be seen at all stages, so the investigation of the stage and visional powers of the case must be noted accurately, and these will be the guide to treatment.

Points to Note.

The following points are to be noted: (i) History (age of onset, hereditary evidence); (ii) class of squint (paralytic or concomitant, in or out); (iii) fixation power of the squinting eye; (iv) range of movement of each eye; (v) vision of each eye; (vi) refraction of each eye; (vii) grade of binocular vision; (viii) angle of squint with and without glasses at a distance (static) and near (dynamic) convergence.

Most squints are painfully obvious to surgeons and parents; some are only apparent (widening or flattening of the bridge of the nose, mongolian eyelids) and others are very difficult to see. A high + or − value of the angle γ is such a case in point. It will be remembered that the line of vision often passes five degrees in and two degrees below the optic axes in hypermetropia (+ angle γ), therefore, in the primary position hypermetropes may appear to diverge ten degrees, myopes with − angle γ appear to converge.

In infants, direct a beam of light on to the eyes and, watching through the spy-hole of the ophthalmoscope, note the position of the reflex in each pupil. Corresponding positions, orthotropia. Note if the deviating eye is able to fix the light correctly. Fixation of first one eye and then the other on an object such as a watch or pen is often fallacious, as large moving objects may be followed by an eye with very defective sight, which only fixes erratically.

Measure the deviation with a Priestley-Smith's tape. In older patients, direct their attention to a spot of light at six metres. Cover first one eye and then the other, and notice if any sharp eye movement occurs as either eye is uncovered. Measure the deviation with a Maddox tangent scale or perimeter. Notice if the deviation increases on near vision (dynamic convergence).

Has the Patient Binocular Vision?

In children under five the four-dot test (Worth) may be successful. The patient has a red glass before the right eye and a green before the left, and is shown a red light dot, two green and a white. A red and white seen = left amblyopia. Two green and a white seen = right amblyopia. A red, two green and white = I or II binocular vision. A double white dot = diplopia.

In others: (*a*) The "friend" test in red and green letters is viewed through a red filter before the right eye and a green filter before the left eye. If the whole word is seen, there is binocular vision; if only "fin", the right eye is suspending vision; if only "red", the left eye is suspending vision. (*b*) Bar-reading test. Ask the patient to read a few sentences in a book, without moving book or head, whilst holding a flat ruler still between the eyes and the book. Try it. Binocular vision is necessary to read the whole line.

What is the Vision of Each Eye?

Worth uses various sized ivory balls for infants to determine the vision of each eye. At six metres he says that a one and a half inch ball = $^6/_{60}$, roughly, and a three-quarter inch ball = $^6/_{24}$. Pennies thrown on the floor form another rough guide. Older children read test types or pictures (Snellen's at six metres).

Refraction.—Retinoscopy (*oculentum atropini* 1% three times a day for three days, or in older children oily hyoscine 0·25% for one hour may be used as a cycloplegic) is essential and a subjective test is attempted. Errors of refraction are fully corrected in accordance with accepted rules, in hypermetropes with convergent squint; slightly under-corrected perhaps in divergent squints, and slightly over-corrected in myopic divergent squints sometimes, if found necessary. If stronger (+2·0 add) spheres are used for children to abolish accommodational convergence; order them as bifocals. These are sometimes tried as a temporary expedient when the child has straight eyes for distance, but squints inwards when reading. I have never used them yet.

Squinters who are emmetropic, with good vision in each eye, are obviously lacking in the fusion sense; this may be congenitally absent. It is possibly a Mendelian inherited factor.

Squinters who are emmetropic, with defective sight in one eye, have congenital amblyopia, a rare condition; the vision is usually around $^6/_{60}$ or $^6/_{36}$, and corresponds to the vision of the retina within ten degrees of the macula. It would appear that the macula is insensitive in these cases. Operate early. Squinters who have errors of refraction of considerable amount in one or both eyes, with defective vision in one eye, are suffering from *amblyopia ex anopsia*, that is, from disuse, and this type of amblyopia may be much greater than congenital amblyopia; it may come on very quickly and is often permanent in degree when first seen. Vision may be of shadows only.

Treatment.

We have now arrived at the most common type of case, that is, convergent concomitant strabismus with hypermetropia, and this is how I treat them at King's College Hospital.

Treatment commences as soon as a squint is found, and all children where there is any history of squint are examined for acuity of vision in each eye, and in doubtful cases refracted under *oculentum atropini* 1%. Where errors of refraction are found in squinters, glasses are prescribed, no matter what the age. Tie-on glasses are used for babies. See the patient again in one month. Many patients are cured by glasses, and ordinary supervision carries them on to full stereoscopic vision.

Many patients are not cured. Order atropine ointment 0·5%, to be applied every morning to the fixing eye. This often insures that the atropined eye is used for distance and the non-atropined eye for near vision. Never order atropine to both eyes in lieu of glasses, as this is a fatal procedure and speedily (three months) may cause irremediable *amblyopia ex anopsia* in many cases (the eye with the greater error of refraction obviously suffers most). The atropine ointment may be ordered less frequently and intervals may be tried.

If no improvement occurs, try occlusion for the fixing eye for one month at a time all day, and watch the vision of the two eyes at each visit for fear of reversing matters. Instruments used to stimulate the vision in amblyopic eyes are called optophores, synoptophores and kaleidoscopes. I consider the every day stimuli of life, food, sharp corners on furniture, or falling (downstairs!) are more penetrating, effective and continuous.

As soon as the squinting eye has reasonable vision, $^{6}/_{12}$, attempts are made to elicit diplopia at a distance by means of lights and coloured filters. A variable prism may be used, although the jerky jump movements of a frame of prisms are more stimulating (this is the principle of the kratometer).

Diplopia, simultaneous perception, Grade I vision. At this point fusion exercises are commenced (see later).

Quite a number of squints will become alternating squints, and these are difficult to deal with; for these Dobson-Hearn spectacles are worth trying. The nasal fields are restricted by the occlusion of the nasal half of each lens and nasal guards placed well into each inner wall of the orbits.

Quite a number of unilateral convergent squints will not straighten and the suppression of vision continues in the squinting eye when both eyes are open. With these I try a non-actinic ruby red clip-on filter to the fixing eye. The vision of this eye is altered and the images of both eyes are then necessary to give a sufficient fullness to the visual impressions of colour and contrast.

If improvement is not setting in before five years of age, all large squints (over 35 degrees) should have a recession operation on the internal rectus muscle of the squinting eye. Treatment after operation continues where it was left off, and frequently fusion training will now be possible. As soon as diplopia (simultaneous perception) has been elicited, correct any hyperphoria present: this is most important, for it has been found that although fusion is possible of images placed on non-corresponding points of the retinæ, it is much easier to fuse images formed on corresponding points. Objects placed on the horopter of Muller, the segment of a circle passing through the nodal points of each eye and the fixation point, stimulate corresponding points on the retinæ. All points outside or inside this circle form images on non-corresponding points.

Fusion Training.

Most people have a slight muscle imbalance or heterophoria for reading at one-third of a metre (33 centimetres); it is found that there is often about 6 △ (prism diopters) of exophoria, but the fusion sense maintains single vision. At 33 centimetres we use three diopters of accommodation and three-metre angles of convergence if orthophoric. Now a one-metre angle of convergence with an interpapillary distance of 60 millimetres is represented by 6 △ of convergence. So three-metre angles = 18 △ of convergence. If, however, we have 6 △ of exophoria at 33 centimetres, we are using only 12 △ of convergence. Hence, for every diopter of accommodation we use 4 △ of convergence, so that an uncorrected hypermetropia of 1·50 for distance would be orthophoric for reading (4 + 4·5 = 18). Fusion is therefore the power an individual possesses of overcoming a muscle imbalance. It may be positive (as in exophoria) or negative (as in esophoria). The fusion power or amplitude is the sum of these two, and it may be measured in prism diopters (△) by a variable prism, the amount which breaks the single image into two.

Convergence is therefore of three kinds: (*a*) tonic or static (+ exophoria, − esophoria), (*b*) accommodational (4 △ to 1 m.a.), (*c*) fusional.

In convergent squints we have a deficiency of negative fusional convergence, and fusion training aims at increasing the fusion amplitude in the negative direction. Accommodational convergence is suspended by + spheres in the training instruments. The various instruments are therefore designed so as to place the images of two similar objects on the macula of each eye in the squinting position and then allow them to be moved slowly apart outwards and inwards. Graduated scales are available to show how extensive these movements are before the fusion breaks down and the picture breaks into two.

Our choice of instruments is extensive, but the following are the best, in my opinion:

1. Worth's amblyoscope (Theodore Hamblin, Limited) (Figure I), from	£2 0 0
2. Pugh orthoptoscope (Figure II)	£38 10 0
3. Maddox cheiroscope, complete with attachments (Figure III)	£11 3 0
4. Sheila Mayou strabismuscope (surgeon's model) (Figure IV)	£15 0 0
5. Sheila Mayou strabismuscope (patient's model)	£8 10 0

6. "Cruise" variable prism stereoscope (Figure V)	£3	17	6
7. Hamblin's drawing stereoscope	£1	8	6
8. Holmes stereoscope, from	£0	12	6
9. British refracting unit, complete with stand (Figure VI)	£112	0	0
10. Hamblin drawing stereoscope with rotatable prisms	£4	5	0
11. Whittington series, junior or senior (for use with Hamblin's drawing stereoscope), per set of 10	£0	18	6
12. "Cruise" series, per set of 20	£0	12	6

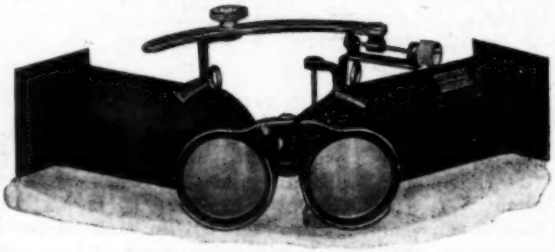

Figure I.
The Worth amblyoscope. (Hamblin.)

This last instrument (number 10) is the best model of all, in my opinion, and combines some of the best points of the "Cruise" stereoscope (variable prisms) with the best point of Whittington's, the drawing vanishing stereograms ("Printator" wax tablets). The model is a reasonable proposition (price) for home training, and the drawing stereograms are extremely efficient in holding the interest of quite young children. The light on

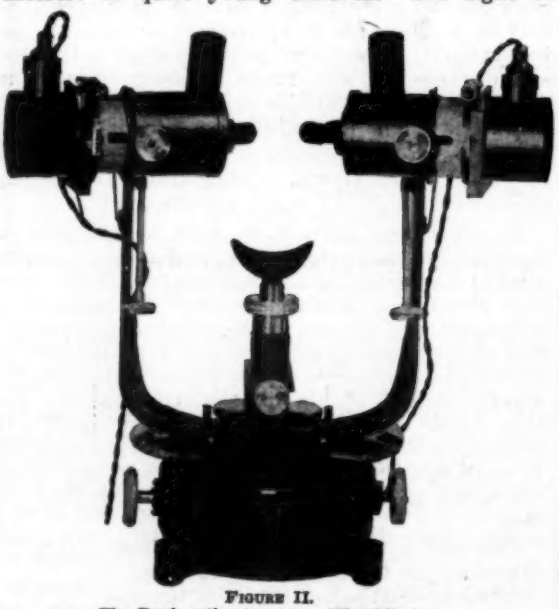

Figure II.
The Pugh orthoptoscope. (Hamblin.)

either side can be intensified, fusion is aided by the prisms, and the drawing holds the interest and assists the cooperation of hand and eye. Left-handed children often have a dominant left eye or defective right eye.

Worth's amblyoscope is quite a handy instrument and children like it. Commence by demonstrating diplopia by Grade I pictures, bird and cage. Slide the bird to and away from the cage to demonstrate the principle involved.

Figure III.
The Maddox cheiroscope.

Continue with Grade II pictures. Part of each picture is common to both and so forms a lock which stimulates fusion. Move the arms to break the pictures each way and measure the fusion amplitude on the scale.

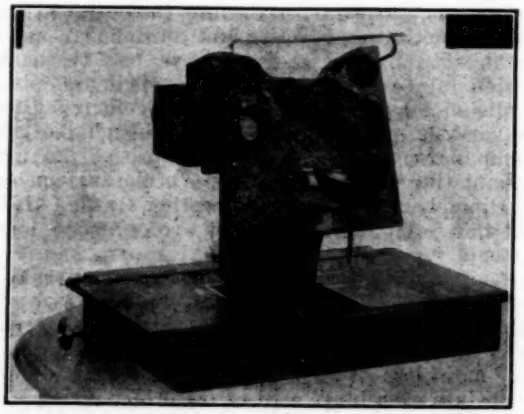

Figure IV.
The Sheila-Mayou strabismuscope.

Later, try Grade III pictures, which require stereoscopic vision (Figures VII and VIII). The Griffith series and the Air Force series are good ones. Changing over the pictures causes a "conversion of relief". The bucket appears to be turned upside down instead of the right way up.

The Maddox cheiroscope allows of actual movements of solid or flat objects by hand, and at the same time they may be drawn by the patient. Non-squinters are trained with the mirror: (i) covered

or (ii) absent. Squinters are trained with the mirror: (iii) oblique for low degrees of squint, (iv) vertical for high degrees of squint. My patients are trained at the Royal Eye Hospital with this instrument.

Figure V.
The Cruise stereoscope and pictures.

The Holmes stereoscope is a simple instrument suitable for home training for squint of less than 20 degrees. Stereogram cards are arranged with varying separatins of the pictures to suit all cases.

The surgeon should commence the fusion training with half-hour sittings as weekly intervals, and then home training is undertaken once or twice daily for fifteen minutes at a time. Whether fusion training is worth the time and expense to patients is still a doubtful point and its value is still of problematical amount. Operation, however, remains the most effective measure in very large squints and neglected cases.

Figure VI.
British refracting unit.

Operations.

Thirty-five degree squints and over require a recession operation on the squinting eye. Aim to reduce the squint by 20 or 25 degrees. Fifteen to twenty degrees of remaining strabismus can easily be corrected by advancement three months later, not sooner. The advancement operation may not be necessary if three to six months' interval is given and fusion training is used.

Recess one millimetre for every five degrees of squint you wish to correct. Never behind the equator; five millimetres bring you to the equator.

Advance two millimetres for every five degrees of squint you wish to correct, taking up muscle to the necessary amount.

Recession of an external rectus muscle is not an effective measure for divergent squints; 2·5 millimetres bring you to the equator. I have never performed tenotomy, as I have never understood the working of this mutilation, which often leads to a divergent squint later in life.

Alternating squints are often best corrected by bilateral advancements.

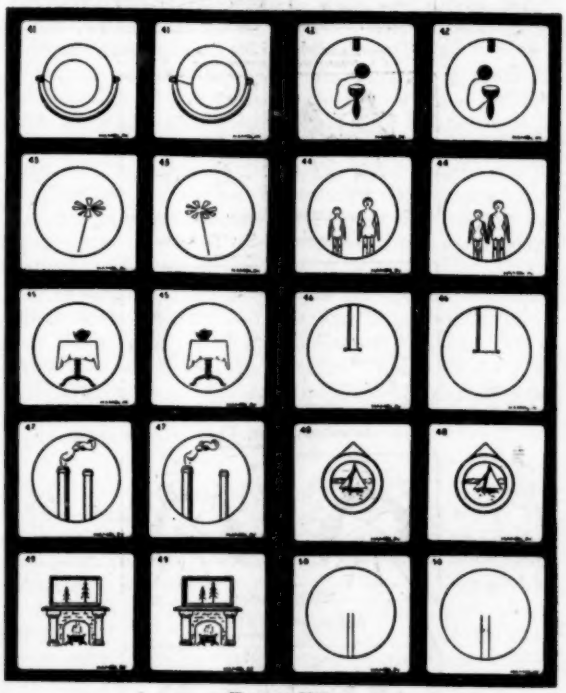

Figure VII.
Amblyoscope pictures. (Griffith series.)

Recession.—In young children general anæsthesia is necessary; in others, cocainize the eye in the usual manner: four drops (4%) four times at four-minute intervals with four single drops of one in 10,000 adrenaline interspersed, and then inject the retrobulbar space with 0·75 cubic centimetre of "Novocain" (2%) with adrenaline (one drop of adrenaline). Wait seven minutes. Incise the conjunctiva vertically eight millimetres to the inner side of the corneo-scleral junction. Incise Tenon's capsule between the superior and internal rectus. Do not separate the muscle from its neighbouring capsule or conjunctiva. Hold the muscle with Worth's strabismus forceps 2·5 millimetres from its insertion. Cut off the insertion close, but not too close to the globe (the sclera is often only of paper thickness beneath the tendon); leave 0·25 to 0·5 millimetre of tendon. Hold the eye by this remaining

tag. Stitch the muscle to the globe with "000" chromicized catgut on single, fully curved, cutting edged (eyeless) needles (number 4 curved needles, Weiss), using three stitches. The needles pass through two-fifths of the scleral thickness in a vertical direction. Take the muscle 0·5 millimetre back from the cut edge, then sclera and then muscle again. Tie the central stitch first and the upper and lower simultaneously with your assistant, using

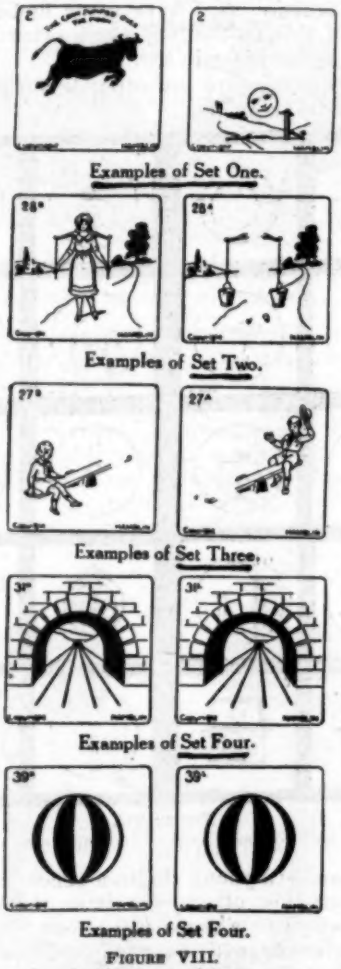

Figure VIII.
Amblyoscope pictures.

surgical knots and three knots to each. Do not splay out the upper and lower stitches on the globe; if one slipped, considerable rotation of the eye might follow. Close the conjunctival wound with two or three number 1 black silk sutures. Instil atropine drops (1%) afterwards and use a pad and bandage for both eyes, if possible, for six to ten days. The eyes are bathed daily and the conjunctival stitches are removed about the fourth day. The risks to the eye are: detached retina, perforation, sepsis to the wound, and loosening of the new attachment; so use patience and sit down to it if necessary.

Advancement.—In doing an advancement operation, make the conjunctival incision vertically four millimetres from the corneo-scleral junction. Pick up the muscle as before and cut off one millimetre from the globe. Stitch to the globe with three sutures of "000" catgut on similar needles. Take conjunctiva, muscle, sclera, muscle, conjunctiva; clear the sclera well of all areolar tissue before suturing. Place the sutures two millimetres from the corneo-scleral junction and pierce the muscle well back behind the clamp forceps. The muscle should not be cleared from the conjunctiva. Close the conjunctiva as before. Do not touch the catgut sutures; when fixing stitches are removed there is a risk of detachment of the muscle insertion.

THE AFTER-TREATMENT OF FRACTURES.

By E. B. Clayton, M.B.,
Director of the Physio-Therapeutic Department, King's College Hospital, London.

Physiotherapy is of great value in the after-treatment of fractures. It shortens the time for recovery of the full use of the limb, and in many cases turns what would have been a poor into a good functional result. It is used with recent fractures to reduce swelling, ease pain and to prevent stiffness of joints. At a later stage it may be required to loosen joints which have become stiff as the result of prolonged immobilization, for persistent œdema, especially with fractures of the lower limb, for pain or for muscular weakness. Then again it may be needed for complications such as nerve injuries, arthritis or ischæmic contraction.

The main essential is movements which must vary from gentle assisted movements in the early stage to strong exercises in the final stage of treatment. With a great number of fractures in children and young adults physiotherapy is unnecessary, the full movements being quickly recovered when the limb is freed from splints. But older patients frequently develop stiffness, which gives far more trouble. This stiffness may appear in joints which have not been injured and may develop gradually while the limb is immobilized. For instance, the shoulder joint often becomes stiff after a Colles's fracture, or the toes after a fracture of the leg. Occasionally every joint in the limb is affected and several months of treatment are required to restore mobility. It is therefore a wise plan to arrange that every joint which is not fixed by the splint is moved through its full range of movement at least once a day from the start.

For the sake of clearness I will divide the after-treatment of fractures into three stages: (i) The stage of rest. The joints which are fixed by the splint are not moved. (ii) The stage of rest and gentle movement. The splint is now removed during treatment and the joints which were fixed are given one gentle movement. (iii) The stage of exercises. This commences when the splint has been discarded. The exercises are used to restore the full function of the limb.

The First Stage.

During the first stage the joints which have not been fixed should be moved through their full range at least once a day to prevent the development of stiffness. Where there is a tendency to stiffness the muscles should be massaged. In certain cases the fixation bands can be removed and massage in the form of *effleurage* can be applied along the limb and over the fracture to reduce swelling and ease pain. This is especially valuable in elderly patients. Heat can also be used for the relief of pain. Radiant heat may be given from a radiant heat bath with carbon filament lamps, from a single lamp, an infra-red ray generator, or an electric fire.

The Second Stage.

With the second stage the splint is removed for massage and movements. The joints which had been fixed are given one movement in each axis. The patient is assisted in performing a gentle active movement, the movement being stopped as soon as any resistance is felt. Where there is any possible danger of displacing the fracture, the bone must be carefully supported throughout the movement. The splint is replaced after the treatment.

It is very difficult to give exact times for the commencement of this second stage. Union should be sufficiently firm for the movements not to disturb the position of the fracture. The date will depend on the severity of the fracture, the time for union to occur, and the skill of the person who performs the movements. With a Colles's fracture, finger, elbow and shoulder movements are practised from the first day. Wrist and pronation and supination movements might be commenced at the end of a fortnight and the splint discarded after three or four weeks. With earlier wrist movements there is a risk of displacing the fracture. Further, they do not seem necessary, since we have found that the great majority of patients obtain a perfect functional result with the above time-table. The ordinary Pott's fracture does not need early physiotherapy. Massage, ankle and toe movements and flat foot exercises, given in the sitting position, can be commenced at the end of five weeks, so that the function of the foot can be restored to some extent before walking is allowed at the end of seven weeks. On the other hand, massage to the whole limb, including the shoulder, and movements for the elbow and joints below should be started as early as possible for an impacted fracture of the surgical neck of the humerus in an elderly patient. Gentle shoulder movements can usually be started after a fortnight. At the end of a month, if there is not much pain, the arm can be carried in a sling outside the clothes.

Limited shoulder movement is the chief complication after a fracture of the clavicle. If the method of fixation allows, shoulder movements could be commenced after about ten days. The movement must take place only in the shoulder joint and not cause any elevation of the acromial end of the clavicle. Rotation movements will be especially useful. The fixation bands can be removed at the end of three weeks.

With fractures of the lower end of the humerus the arm is commonly fixed with the elbow flexed. This position is maintained for three weeks. Then active elbow movements are allowed. The special exercises mentioned below for a stiff elbow should not be used until after eight weeks. These precautions are taken because there is a danger that *myositis ossificans* may develop in the *brachialis anticus* muscle. This would be encouraged by early vigorous movements.

A fracture of the olecranon without displacement would probably be treated with the elbow on a splint for about three weeks. The difficulty in after-treatment is to restore flexion. After practice in gentle active elbow flexion the arm is fixed in the position of the greatest flexion attained, either with an adjustable internal angular splint or in a sling. This is continued until full flexion is reached. Vigorous exercises to loosen the joint should not be used before six weeks. A comminuted fracture of the patella without displacement is also rested for about three weeks before gentle knee movements are started.

It is impossible to give dates for the movement of joints with fractures of the shafts of bones, since they vary very much in individual cases. It is important to move the patella from side to side with fractures of the femur, since it may become adherent to the femur and so make the restoration of knee movement later more difficult.

The Third Stage.

The stage of exercises commences when the splints have been discarded. The aim is to restore full mobility to the joints and to redevelop wasted muscles. Massage is continued in the early part of this stage to improve the circulation of blood through the muscles. At first the exercises will be merely simple, free, active movements of the joints in their different axes, or, if necessary, movements helped by the masseuse. Later, stronger exercises are used and, if the joints remain stiff, forcible manipulations. Occasionally manipulations under an anæsthetic are required before full function is restored. Where there is definite weakness of muscles the surging faradic current can be applied to cause muscle contractions; when used in conjunction with exercises, it seems to hasten the recovery of wasted muscles.

Flat Foot Exercises.

Flat foot often develops after fractures of the lower limb. It is therefore important to start a course of flat foot exercises before walking is allowed. The following exercises would be suitable.

For the Anterior Transverse Arch. (1) Sitting toe flexion and extension at the metatarso-phalangeal joints. Rest the foot as far as these joints on a thick book, so that flexion may take place there and not in the interphalangeal joints.

(2) Sitting, draw up the anterior transverse arch and try to grasp some object, such as a pencil placed longitudinally under the arch. Then relax and repeat.

For the Longitudinal Arch. (1) Sitting toe flexion, foot inversion, and ankle dorsiflexion. At first these can be practised separately, later together.

(2) Sitting, draw up the longitudinal and transverse arches. Then relax and repeat. Each exercise should be performed thirty to fifty times, about three times a day. When the patient walks he should be instructed to point the feet forwards or slightly inwards. It is also advisable to wedge the heel of the shoe a quarter of an inch on the inner side. In addition, massage and the faradic current should be employed if there is marked weakness of the muscles or pain in the arch.

The faradic current can be conveniently applied with the foot in a bath, the water covering the toes, but not the instep. The current is surged by pushing in and out the iron core or the secondary coil, according to the make of apparatus. For the longitudinal arch the electrodes are placed at the front and the back of the foot, and for the transverse arch on each side. In addition, surging faradism should be applied to *tibialis anticus, tibialis posticus* and *flexor longus digitorum* muscles by placing a small electrode on their motor points. The circuit can be completed with an indifferent electrode placed above the knee.

General Remarks on Exercises for Muscles.

Exercises to develop weak muscles are simple to arrange, provided that the actions of the muscles concerned are known. For instance, the main action of the deltoid muscle is abduction of the arm from the side. The exercise can be regulated by arranging that a certain weight is raised a definite number of times. If the circumflex nerve has been injured and the deltoid muscle is very weak, it may be impossible to raise the arm actively from the side. The movement should then be performed in the horizontal position, where the weight of the arm is not raised against gravity. With the lower limb the body weight can be used as a resistance for the hip and knee extensors and calf muscles, as in the well known exercise, "standing, heels raised, knees bend". A grip dumb-bell can be used to tighten the muscles of the arm and to resist the movement with the opposing muscle. With practice this self-resistance can also be used for leg muscles. Personally, with an intelligent patient, I prefer these self-resisted exercises to those resisted by a weight, and use them for the leg as well as the arm.

Special exercises should be arranged for stiff joints, which the patient can practise several times a day, in addition to manipulations from the medical practitioner or masseuse. Manipulations and these special exercises should be discontinued if the joint gradually becomes stiffer, or if they cause pain which continues for several hours. These are indications that there is still inflammation present, and the joint should therefore be rested. The following are some of the special exercises which can be used for stiff joints.

The Shoulder Joint.

1. *Pulley Exercise.*—A pulley is fixed overhead and a rope passed over it, one end being grasped in each hand. By pulling down with the sound arm the stiff arm is raised. At first the exercise is performed facing, later with the back towards, the pulley.

2. *Towel Exercise.*—A towel or rope is held behind the back with one hand behind the shoulder and the other behind the waist. By pulling with the sound arm the stiff arm is drawn further behind the back. With the stiff arm above, the movement will be abduction and external rotation, and below internal rotation and extension.

The Elbow Joint.

1. *For Extension.*—Standing, pass a rope under the sole of the shoe in front of the heel. Grasp one end in each hand and extend the elbow by pulling upwards with the sound arm.

2. *For Extension.*—Strap the upper arm to a table with the forearm from the elbow projecting over the edge. Fix a pulley below.[1] Grasp the ends of the rope with the hands as before. By pulling with the sound arm the elbow will be extended. This exercise is better than exercise 1 (that described for extension) for obtaining the last few degrees of extension. For flexion fix the pulley behind the shoulder.[1] The towel exercise, mentioned under exercises for the shoulder joint, can be used for flexion if the stiff arm is above.

Radio-Ulnar Joints.

The elbow is flexed to a right angle and held closely to the side throughout the exercise. One end of a heavy stick is held in the hand. Pronation and supination movements are performed actively, the weight of the stick, as it swings over, assisting the movement.

The Wrist Joint.

Extension is the most important movement in this joint, since the wrist must be extended for a firm grip with the fingers. The following exercises can be used.

1. Hold the hands in front of the body with the palmar surfaces of the hands and fingers in contact. Then, pressing the hands tightly together, draw them backwards towards the chest. As the hands approach the chest the wrists will be extended.

2. Place one hand flat on a table and, pressing the heel of the hand down with the other hand, raise the elbow, thus extending the wrist.

Finger Joints.

1. With the hand in hot water, grip and squeeze a sponge.

[1] With these pulley exercises the pulley should be fixed at such a height that the pull of the rope will be perpendicular to the limb when the limit of active movement has been reached.

2. Hold a walking stick vertically, with the crook downwards, and grip the stick with the fingers. If the fingers cannot close sufficiently to grip the stick, wrap a bandage round the stick to make it thicker. A weight can be attached to the crook to insure a tight grip. Progress by using a thinner stick.

3. Press the back of a book between the fingers to force them apart, using a thick book for the thumb and index finger.

The Knee Joint.

The following exercises can be used to improve flexion.

1. Standing, advance the injured leg, placing the toes against a wall. Then force the knees forwards with a lunging movement. The same exercise would also be of value for developing dorsiflexion in the ankle joint.

2. When the knee can be flexed to a right angle, further flexion is best obtained by flexing and extending the knees in the kneeling position, the body weight being used to help flexion.

There is often a certain amount of œdema round the ankle when the patient commences to walk after a fracture of the lower limb. Commonly this clears up quickly with exercises and use of the leg. But sometimes the œdema is more severe and persistent. Then a bandage should be worn from the foot to the knee. Massage and exercises are employed to improve the circulation and reduce the swelling.

Sinusoidal current leg baths are also of value for stimulating the skin and improving the circulation.

Radiant heat, diathermy, or the constant galvanic current can be used for the pain on movement which often persists for several weeks with fractures into joints.

Conclusion.

In conclusion I would point out that I have taken movements and exercises in more detail than the other parts of the after-treatment of fractures because I feel that many general practitioners have no fully trained masseuse available with experience in fracture work. They must rely on themselves and on the patient's friends for the after-treatment. I would emphasize the importance of regular movements of joints not fixed by the splint, and of carefully arranged exercises when the splints have been discarded.

SOME POINTS IN THE TREATMENT OF CARDIAC DISORDERS.

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In the treatment of patients with cardio-vascular disease emergencies are apt to arise which call for prompt and efficient handling. Most of these are in the nature of more or less sudden failure of the heart to maintain the circulation.

Heart failure may arise in a variety of ways. On the one hand, the variations in the rate of the heart from different causes may lead to a breakdown; on the other hand, deficiency in the contractile power of the ventricle may tend to bring about failure of the circulation, although the rate of the heart may not be materially altered.

The rate of the circulation is maintained by the propulsive power of the left ventricle. On this depends the supply of oxygen to the vital centres. One of the most dramatic and grave forms of cardiac breakdown is characterized by an acute paroxysm of dyspnœa, which is commonly spoken of as cardiac asthma. It must be borne in mind that this condition has nothing whatever to do with bronchial asthma. An understanding of the mechanism underlying these attacks indicates the correct line of treatment. An acute attack of dyspnœa when at rest is likely to occur when the respiratory centre becomes sensible of a gross diminution in its oxygen supply. These attacks frequently occur during sleep. The patient awakes breathless, and in a state of agitation as a rule. His aspect is pale; he is restless, perspires, and gasps for breath, with a long wheezing expiration. This prolonged expiration sometimes suggests bronchial asthma, but it is easy to note that the entry of the air into the lungs is not impeded. When the attack is at its height the blood pressure often rises considerably. The increased ventilation leads to an increase in the return of blood to the heart, and the pulse rate rises, and the heart thus becomes overloaded.

It is thus apparent that a weakened and defective left ventricle is submitted to the strain of extra work.

The cause of these attacks frequently is that the patient has done too much previously. The disturbance may follow on quite soon after overexertion, or may be delayed and occur only after an interval when the patient is at rest.

In many instances patients with high blood pressure suffer from this disorder, but this is by no means constant.

It is necessary, where possible, to relieve the strain on the left ventricle. The condition is one of great gravity, for a further increase in the inefficiency of the left side of the heart will lead to accumulation of blood in the lungs and consequent acute pulmonary œdema. A dry cough and a few crepitations at the bases are warnings that this is likely to occur.

In order to reduce the irritability of the respiratory centre and to calm the patient's anxiety, an injection of morphine, 0·015 gramme (a quarter of a grain), should be given at once. This is the most effective and rapid means of allowing the left ventricle to recover. At the same time it may be desirable to give along with it some atropine to check bronchial secretion and help to allay the cough; 0·86 milligramme (one seventy-fifth of a grain) may be given hypodermically.

Hyperpietics tend to have a blood pressure increased above their normal in this condition, and for them amyl nitrite, by inhalation, may be most valuable, but this drug should, of course, not be given if the tension of the pulse is low.

These measures will help to take the load off the heart.

The next indication is to provide a stimulant for defective contraction. An intravenous injection of strophanthin is the quickest and most effective remedy; 0·43 milligramme (one one-hundred-and-fiftieth of a grain) may be given. The French preparation of strophanthin, "Ouabaine", is very effective. One-quarter of a milligramme (one two-hundred-and-fortieth of a grain) may be given intravenously.

Such measures as these will usually relieve the patient of his symptoms, and further attacks must be avoided, if possible, by more satisfactory regulation of his activities.

Auricular fibrillation and flutter, and paroxysmal tachycardia, all cause sudden and pronounced increase in the rate of the heart. These disturbances are often very well tolerated, but in patients whose hearts are diseased, failure may rapidly supervene. In the case of auricular fibrillation and flutter it is necessary to bring about control of the rate of the heart by digitalis as soon as possible. An intravenous injection of strophanthin should be given at once and the patient then brought under digitalis control as soon as possible, having in consideration his body weight. It is convenient to remember that 0·04 mil per kilogram (one and a half minims per pound) of body weight will digitalize the patient, if given all at once; but this is not a desirable thing to do. Half the dose may be given at once, followed by one-third in six hours, and the remainder perhaps, two one-sixths, at six-hour intervals. In eight to ten hours a definite decrease in the rate of the heart will usually have taken place. Morphine may always be given at the onset to allay dyspnœa and anxiety.

In the case of paroxysmal tachycardia, whether auricular or ventricular, the digitalis group have no definite effect. The drug of choice in these cases is quinidine; 0·3 gramme (five grains) may be given by mouth in an acid mixture, or in a cachet, followed by further similar doses at six-hourly intervals. Of the hydrochloride 0·06 or 0·12 gramme (one or two grains) may be given intravenously. Ocular compression or pressure on the vagus is rarely effective. An intravenous injection of strophanthin may sometimes appear to have an effect, but with the capricious termination of these attacks its efficacy is difficult to determine.

Hitherto our considerations have been mainly confined to failure on the left side of the heart. These acute symptoms may be present in some cases with little or none of the severe venous engorgement which is the characteristic indication of inefficiency of the right ventricle. Distended jugular veins extending up to the sterno-mastoid when the patient is sitting at an angle of forty-five degrees or more, call for the consideration of venesection. The external jugular vein should be emptied by parting the fingers pressed upon it and its filling from below observed. The amount of pulsation in it may not be striking if the distension is considerable. A grossly distended vein indicates that blood should be let, and this is best done by a simple longitudinal incision with a sharp scalpel of the media-cephalic vein at the right elbow. This vein may be rendered more prominent by a tourniquet, but it is often already considerably distended. Anything from 300 to 600 cubic centimetres (ten to twenty ounces) of blood may be conveniently withdrawn. Light pressure with the arm elevated soon stops the bleeding.

It must be remembered that venesection may prove ineffective if it is delayed too long. Sometimes a patient has become very cyanosed; the heart is very feeble and the circulation is so poor that little or no blood is obtained when the vein is incised. In these cases venesection has been delayed too long.

Sometimes a strong retrograde pulsation is seen in the external jugular vein. This is associated with great hypertrophy of the right ventricle and relative incompetence of the tricuspid valve. It is not an indication for venesection unless the vein is permanently tense and distended as well as pulsating. In these patients the state of the venous pressure can conveniently be noted by holding the arm outstretched at various levels compared with the level of the right auricle, and it can be seen how soon the vein becomes empty when the arm is elevated above this point.

Venesection often gives rapid relief to the feeling of oppression and tightness of the chest and also, to a certain extent, to the dyspnœa. The relief it brings to the heart may be very considerable, but the prognosis is bad if the heart cannot take advantage of this relief for long and if the venous distension returns again so that further bleeding is needed.

Adrenaline is of value in stimulating the heart in three conditions: In the somewhat rare Stokes-Adams disease the liability to ventricular standstill is often averted by 0·3 mil (five minims) given hypodermically, the effect of which may last some hours. I find it also useful, in small doses of 0·12 to 0·18 mil (two to three minims) hypodermically, when the contraction of the heart is feeble and the blood pressure low. Sometimes this combination is found in persons with gross myocardial disease, but in other cases such physical signs are not present and syncope is found to occur in an otherwise normal heart. In these cases the blood pressure is low, but at the same time there is often profound slowing of the heart rate owing to the effect on the vagus. These patients are not suitable for treatment by the digitalis group, as their stimulating effect on the vagus would tend to make the condition worse. The pulse being slow—usually about 40 to 50—and sometimes slightly irregular, atropine is called for to paralyse the vagus, and a combina-

tion of 0·86 milligramme (one seventy-fifth of a grain) of atropine hypodermically with 0·3 mil (five minims) of one in a thousand adrenaline is a very quick and effective remedy.

Oxygen in the relief of cyanosis due to stasis in the capillaries following slowing of the circulatory rate can be of value only if introduced into the lungs in large quantities and if at the same time it can pass through the lungs into the blood stream. It is unfortunately only too apparent that patients who suffer from failure of the right side of the heart following diseases of the lungs, such as emphysema or extensive fibrosis, are but little relieved by the administration of oxygen; but where the lungs are reasonably healthy some improvement is usually observed in the cyanosis if the oxygen be introduced through a nasal catheter. This is quite the simplest and most effective way of increasing the patient's oxygen supply. The gas should pass at the rate of about one bubble per second. The nasal catheter should rest just short of the back of the nose and may be smeared with a little "Novocain" and vaseline if it irritates the patient; most patients, however, stand it very well.

A BRIEF SURVEY OF TRANS-URETHRAL TECHNIQUE FOR BLADDER NECK OBSTRUCTIONS IN THE PROSTATIC SUBJECT.

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Amongst the recent advances in urology none has attracted greater attention than per-urethral removal of bladder neck obstructions. Though the majority of such obstructions are of prostatic musculo-glandular tissue, there are others to be considered, as for example, the "prostatic bar", a projection best identified by cysto-urethroscopic examination and here seen as an elevation behind the internal urinary meatus. The bar consists of fibrous tissue only and indicates, in all probability, a preexisting inflammation. Additional obstructions are post-operative fibrous tissue formation, tags of prostatic tissue and capsule (post-prostatectomy) and, finally, prostate carcinoma.

Up to the time of Edwin Beer's epoch-making innovation of endoscopic diathermy the method of elimination of some of the forms of obstruction was either by means of a punch or by a galvano-cautery, originally performed as a "blind operation", but later, with the advent of the posterior urethroscope, guided by a degree of accuracy. Until endoscopic diathermy was introduced precision could never be claimed for two main reasons: (i) hæmorrhage, from punching, obscured visibility, and (ii) the inflammatory reaction and resulting fibrosis from the galvano-cautery could not be controlled. Since diathermy introduction this is changed; the high frequency current coagulates tissue and thromboses vessels, so that a bloodless field can be produced and the subsequent fibrous tissue formation is less dense and contracting than after the galvano-cautery. Bladder neck obstructions, if not too large, can be removed by coagulation necrosis so produced in the same way as a villous papilloma within the bladder may be, but this method, though simple in technique, and safe, has the disadvantages of delayed action, the necrosed tissue requiring a certain length of time to be cast off. This suggests incomplete removal of obstruction for, perhaps, several days or weeks, but actually in practice we find this is not so, as the obstructing elements are disintegrated and softened and allow the passage of a fair stream of urine. The presence of necrosed tissue will predispose to bladder sepsis, but this sepsis is usually of a harmless nature, "silent sepsis" it may be called, as it is usually accompanied by no other symptoms than pyuria. Occasionally epididymitis develops, and sometimes rigors and transitory pain suggestive of an upper urinary tract involvement have been noted, but for the most part such symptoms are absent.

Introduction of the endothermy cutting current has stimulated a new phase in the development of per-urethral methods, for, with the aid of the current, a wire loop may be so activated that it will cut as cleanly through a prostatic projection (even a fibrous or carcinomatous projection) as a chandler's wire through a piece of soap. Thus strips of prostate may be reamed off until the bladder base shelves smoothly into the prostatic urethra, by the so-called "resection" method. The operation is performed under accurate observation. Bleeding vessels laid open by the cutting wire (for this cuts rather than coagulates and so does not thrombose vessels) are subsequently sealed off by contact with a diathermy electrode substituted for the cutting wire.

Punches of various descriptions have come and gone. As already mentioned, before diathermy introduction there was no sound way of checking hæmorrhage, and disasters, as a consequence, brought the method into disrepute. Diathermy was a welcome auxiliary and punches may be now described in two groups, those in which (i) diathermy coagulation precedes punching, and (ii) punching precedes diathermy. In the first the tissue to be removed is first rendered anæmic, then punched away. In the second it is punched as well as the hæmorrhage that occurs will permit, and the bleeding vessels are then coagulated. This method is carried out by Caulk, who records its use in over 800 cases with a mortality of under 1%.

Another method, that of Collings, employs a diathermy knife, a blunt, flat-bladed knife, which is merely a modified diathermy electrode, and this serves either to incise vertically or to cut out a wedge-shaped piece of a projection. It has its best application in median lobe or bar obstructions.

The average urologist has neither time nor opportunity of exploring all these methods. The technique of each is somewhat intricate, and it is wiser to

concentrate upon one or two. The personal experience of the writer led him away from the punch. Hæmorrhage was the bugbear; it either occurred at the operation or subsequently, and was the method used in two of his fatal cases (out of a total mortality of three in six and a half years' practice of all methods). Further, experiments upon the cadaver showed how dangerously close to the ureters the scope of the punch might encroach, and though in no case was the ureter orifice actually involved, yet it seemed possible that similar operations performed upon the living might produce an inflammatory œdema and predispose to ascending infection, either by the ureter lumen or the periureteral lymphatics.

Thus simple electro-coagulation and resection appear to be the methods of choice, as presenting techniques which are more accurate and less disturbing to the patient immediately and subsequently.

Indications.

In deciding the indications for per-urethral treatment, it is unnecessary here to discriminate between the various forms the operation may take. We are only concerned with the indications as they are governed by: (*a*) the type of glandular obstruction, (*b*) the subject for operation.

(*a*) *The Type of Glandular Obstruction.*—Intravesical projections of lateral and middle lobes, simple or combined, the median bar, fibrotic glands, post-prostatectomy obstruction, and some cases of carcinoma (of low malignancy).

(*b*) *The Subject for Operation.*—The mild prostatic with annoying but not dangerous symptoms, whose occupation prevents his sacrificing time for the major operation, subjects of cardio-vascular and respiratory disease; nerve disease, for example, mental defectives (of a major or minor grade), and in those patients in whom it is questionable if the dysuria is due to a neuro-muscular defect (for example, tabetic bladder) or to a cystoscopically visible intravesical projection; fat, flabby, constipated patients liable to post-operative ileus. Those also suffering from vesical papilloma (suitable for fulguration, and calculi suitable for litholapaxy) and those in whom a colostomy has been established likewise come within the scope.

Preliminary Treatment.

All those investigations and preparations preliminary to prostatectomy are just as important before carrying out per-urethral methods. Further, a most accurate visual investigation by cystoscope and posterior urethroscope must be undertaken, not only to check that the obstruction is of a suitable nature, but to realize the existence of additional pathological lesions.

The operation is not complete until all bleeding has been checked by diathermy coagulation, whatever may have been the method employed. A catheter is tied in and retained for three or four days, during which time the bladder is washed out twice or thrice daily, preferably with one in 12,000 silver nitrate solution.

In many cases, especially where there is severe sepsis or deficient renal function, a preliminary cystotomy is performed. This actually renders the operation easier (for the washing is more efficiently carried out and a clearer atmosphere for working in is obtained) and safer (for reaction and septic absorption are both largely prevented in this way). A severe grade of sepsis, especially if associated with toxæmic symptoms, indicates a longer pre-operative drainage in the hope of establishing an immunization, with less likelihood of upper tract sepsis.

Technique.

The details of technique of individual operations can be best studied from monographs by Caulk, Davis, McCarthy, Stern, Ryall and others; it is not intended to extend the scope of this brief survey to include them.

For those commencing this work and who are not too familiar with visualization of the bladder neck, bladder base and posterior urethra, the coagulation by transfusion of intravesical projecting lobes effected with electrodes passed through a Wolf's cysto-urethroscope, or one of similar design, is recommended as being the safest introduction to the method of per-urethral technique. The electrodes should possess stiffer shafts than those usually used for coagulating papillomata, and the metal ends should be slightly longer and pointed.

When acquaintance with common appearances and a degree of confidence have been gained, careful attempts may be made in the use of the McCarthy resectome. In this method it cannot be over-stressed that too much must not be attempted at one sitting. It is far safer to resort to repeat operations in all except the cases with quite small projections. The operation may be apparently trivial in technique, but the consequences may be serious, especially in view of the poor material upon which the surgeon is working. The additional burden of the anæsthetic to be borne by the patient must not be lost sight of. Conservatism is the secret of success. It is no matter for reproach if two or more operations have to be performed in one subject.

It is not to be assumed that per-urethral methods have, in the opinion of the writer, displaced, or are likely to displace, the well established operation of suprapubic prostatectomy. This is emphasized from the fact that through a period of nearly seven years (dated from the commencement of a serious contemplation of these methods) rather under 10% of all prostates seen have been recognized as coming within the scope of endoscopic technique. The patients so treated have, for the most part, been those who could never have been steered with safety through the hazardous paths of prostatectomy and its searching convalescence. There were a few patients who refused, point-blank, a "cutting operation", but were willing to submit themselves to the operation from within.

Analysis of Patients Treated.

Of the 76 patients treated, 21 were over 70, one was 84 and one 90 years of age. Twenty-three patients had had acute retention, eight had retention and overflow, and four had bladder distensions of, in three, 1,800 cubic centimetres (60 ounces) and, in one, 1,500 cubic centimetres (50 ounces). Three cases of carcinoma were treated.

On account of sepsis or deficient renal function in fifteen cases it was necessary to carry out preliminary cystotomy, preserving in some cases drainage for as long as nine months.

As to the methods used, in fifty-three cases electro-coagulation alone was employed; in fourteen, resection by the McCarthy method. In the remainder, diathermy punching, or a combination of electro-coagulation (by transfixing) with punching or resection, was resorted to.

Post-operative complications have been relatively less frequent where electro-coagulation has been employed; punch methods have caused most concern; in fact, two of the three fatal cases in the writer's series followed this technique. Immediate post-operative hæmorrhage occurred only after punch methods and resection (one case). As to post-operative sepsis, the writer has seen little difference in its incidence, whether electro-coagulation or resection has been employed.

Of the three deaths, two occurred in uræmic patients early in the series. One patient was over eighty years of age; the other also suffered from carcinoma of the colon and had recently had colostomy performed for acute obstruction. The third death resulted from post-operative pneumonia and empyema.

In two cases, also early in the writer's series, when, so to speak, nibbling at these methods, his courage failed him to persevere and, as both were patients who were anxious to be put right quickly once and for all, he carried out prostatectomy after having in one previously used electro-coagulation and in the other a punch. Both patients had large prostatic projections and would probably have required several per-urethral treatments. In neither case did the suprapubic operation present difficulties and both patients enjoyed normal convalescence.

Complication.

One complication which may give rise to delayed return of normal function is bladder atony, met with in those patients who have suffered from great distension of the bladder, often through a period of years, before seeking advice. This atony, of course, is not a complication of the operation, but of the condition which preceded it. The atonic bladder recovers far more quickly when rested by cystotomy drainage than in any other way. Where cystotomy has not been done in the class of case referred to, and the original residual urine was anything from one to three pints, it may be necessary to give relief, for a time, by a degree of catheter life, as the case demands. In five of the writer's cases this has been necessary and the result of a residuum approaching zero has proved the truth of the contention.

Duration of Effect.

As to the duration of effect, frequent hospital follow-ups and answers to *questionnaire* letters provide most satisfactory information. Especially is this so in regard to electro-coagulation, for nearly seven years have passed since the commencement of the series. The first patient (January, 1927) had had two attacks of acute retention, and normally a residuum of fourteen ounces was found. No treatment has been necessary since his first coagulation, and today he completely empties his bladder. Experiences with resection are naturally much shorter, and time alone can prove the permanency of the method.

DELAY IN LABOUR.

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It is not possible to define the limits within which the duration of labour may be considered normal. A rough average length of time for its successive stages can be stated, and this will vary in different races and countries. In the British the average duration of labour in a *primigravida* is eighteen to twenty hours, that is, first stage fifteen to eighteen hours, second stage two hours, third stage half an hour. Among *multiparæ* there is more variation, but an average is eight to twelve hours, first stage up to eleven hours, second and third stages half an hour each.

The duration of labour may exceed this average many times or be completed in a considerably shorter time without harm to either mother or child. The only occasions on which labour can be said to be abnormally prolonged are when the prolongation is sufficient to produce signs of maternal or fœtal distress.

If labour is regarded from the mechanical point of view, delay may be due to the following causes:

(i) Weakness of the forces of labour: uterine inertia, uterine exhaustion.

(ii) Irregular action of the forces of labour: contraction ring *et cetera.*

(iii) Obstruction to the advance of the child: obstructed labour.

Weakness of the Forces of Labour.
Uterine Inertia.

Uterine inertia is a condition in which, as labour progresses, the uterine contractions do not increase in frequency and in strength as they do in normal labour. In consequence, dilatation of the cervix and advance of the child are very slow. This condition may be present from the onset of labour and persist until delivery, and though it prolongs labour it usually has no ill effect on the child, but the mother tends to become tired out, depending on

the duration of the labour. When weak and infrequent pains occur in this way from the onset of and throughout the labour, the condition is spoken of as primary uterine inertia.

When the pains begin normally, but after a varying time cease altogether, a condition which is associated with a weak uterine muscle and is more often seen in *multiparæ*, the uterus is said to be in a state of secondary uterine inertia or uterine exhaustion. It has been a time-honoured custom to describe uterine inertia as primary and secondary, resulting in confusion both in theory and in practice; of late years the terms "sluggish uterus" and "exhausted uterus" have been used to replace them. In this article I propose to reserve the term "uterine inertia" for the condition previously described as primary uterine inertia, and confusion will be avoided by my strict adherence to the term "uterine exhaustion" in place of secondary uterine inertia.

Uterine inertia is the commonest cause of prolonged labour, yet its causation is often obscure, and little definite is known of it. It is impossible to foretell the behaviour of any individual uterus in labour; the uterus of the healthy *primipara* is as likely to suffer from uterine inertia as that of the *multipara*, worn out by numerous pregnancies in quick succession. This condition occurs in cases of overdistension of the uterus from twins or hydramnios, in cases of early rupture of the membranes, in those patients allowed to have a distended bladder or lower bowel. Fatigue and emotion are not infrequent causes.

Inertia has its maximum effect in the first stage, but will continue during the second, so that this stage lasts many hours or, indeed, the uterus may fail to push the child past the last resistance, the *levator ani* muscles.

Uterine inertia is diagnosed by observing the nature of the pains and verifying the absence of any sign of obstruction which could, if it were present, be a cause of similar delay. The patient is often restless and tired; the uterus is felt to be soft, the fœtus is easily made out, the fœtal heart is usually normal and the head sunk in the pelvis; when the uterus does produce contractions they are short and painful, in addition to being infrequent. On vaginal examination there is a varying amount of dilatation of the cervix, and the membranes may or may not be ruptured. When the uterus contracts, the finger in the vagina will feel the head pressed down a slight amount. There is usually an absence of such conditions as an œdematous anterior lip of the cervix or a marked *caput succedaneum*, both being some slight indication of difficulty in descent of the head.

Treatment.

Being satisfied that there is an absence of obstruction, treatment of the inertia should be carried out, and this varies with the condition of the patient and the stage of labour. The importance of keeping the patient as fit as possible cannot be over-emphasized. In a labour of this type, which may last three or four days, during which the patient does not sleep, drugs must be given to provide sleep, also food to keep up her strength, if she can swallow it. If vomiting is a feature, as it not infrequently is, then glucose solution given *per rectum* or subcutaneously at regular intervals has an excellent tonic effect. The sedative drugs which are used vary from chloral hydrate, 1·8 grammes (30 grains), and potassium bromide, 2·4 grammes (40 grains), given together by mouth, usually in the early stages, to morphine, 0·015 to 0·02 gramme (one-quarter to one-third of a grain), by hypodermic injection. These are the two drugs most frequently used, but others that may be prescribed are: Hyoscine, 0·4 milligramme (one one-hundred-and-fiftieth of a grain), by hypodermic injection, followed after two to four hours' interval by further doses of 0·15 milligramme (one four-hundred-and-fiftieth of a grain); "Nembutal", 0·18 gramme (three grains), and chloral hydrate, 1·8 grammes (30 grains), followed at two-hourly intervals by "Nembutal", 0·09 gramme (one and a half grains), and chloral hydrate, 1·8 grammes (30 grains). If efficient sleep is obtained, the patient is much improved by it, and the uterine action tends to improve.

The opposite type of treatment can be carried out if the patient is in a good condition, namely, to attempt to increase the uterine contractions by: emptying the bladder, giving castor oil, 60 cubic centimetres (two ounces), followed in an hour and a half by a large hot enema and a hot bath. Quinine sulphate can be given by mouth at the same time, a total amount of 1·8 grammes (thirty grains) in four doses spread over six hours. The above treatment is safe at any stage of labour, as it never produces tonic contraction of the uterus, whereas other drugs, which are more certain and more efficient in their action, are liable to do this and should be given only under certain conditions. Pituitary extract is the most effective drug in stimulating uterine contraction. It is now standardized to contain ten units per cubic centimetre, and two units are usually a safe dose, that is, a dose that does not produce spasm of the uterine muscle. This drug should be reserved for use during the second stage of labour, as serious injury to both mother and child may occur if it is used in the early part of the first stage.

When the uterine action has succeeded in producing full dilatation of the cervix in a patient suffering from uterine inertia, then, and then only, can forceps delivery be carried out as a means of assisting the weak uterine contractions and ending the long and tedious labour. It is usually found that from the help given by the forceps the uterus is stimulated to achieve quite good contractions. Delivery by forceps is the correct treatment of every case of uterine inertia, once the cervical dilatation is complete. This saves the patient many hours of dire discomfort, during which time she becomes more and more exhausted, and in many cases it

serves to carry out delivery before the uterus is so weakened that *post partum* hæmorrhage becomes a probable, rather than a possible, danger of the third stage.

Uterine Exhaustion.

Uterine exhaustion differs from inertia as mentioned above by a complete absence of contractions. The two conditions inertia and exhaustion are not necessarily related, and the uterus exhibiting inertia does not pass on to exhaustion. It is said to occur more often in *multiparæ* who have had many children, rather than in *primiparæ*. It may also arise in the course of a long and difficult labour, in which the uterus is tired out by its frequent, strenuous, but ineffective efforts to expel the child, with resulting exhaustion, muscular and nervous, of the mother, either before or after expulsion of the child or placenta.

Uterine exhaustion is very rare in a healthy patient, but it may arise in the course of normal labour which began with contractions of the ordinary type. It is characterized by complete cessation of uterine activity, the contractions ceasing entirely and the presenting part being freely movable, owing to relaxation of and the absence of retraction in the uterine muscle. There is no danger to the mother and child in the first and second stages from the uterine exhaustion, but in the third stage severe hæmorrhage will result if any separation of the placenta occurs.

Treatment.

The treatment is to maintain the patient's strength, obtain sleep for her, and wait for the uterus to contract again. Delivery in the absence of retraction of the uterine muscle is certain to be followed by dangerous, if not fatal, *post partum* hæmorrhage.

Irregular Uterine Action.

The action of the uterus in labour is sometimes described as "colicky", a condition in which irregular and frequent contractions of short duration occur. It more often occurs in *primigravidæ* than *multiparæ*, and denotes an increased irritability of the nerve supply of the uterus. It is usually relieved by morphine, 0·01 gramme (one-sixth of a grain), given subcutaneously.

Contraction Ring.

A localized spasm of the circular muscle fibres of the uterus is occasionally met with, producing the "contraction ring". It usually occurs around a groove in the outline of the child, most commonly the neck, and in vertex presentations this corresponds with the upper part of the lower uterine segment. The condition is of importance, as it produces marked delay in delivery. The spasm usually develops early in the first stage or even at the onset of labour; it is less commonly seen in the second stage. The condition may occur for the first time during the third stage of labour, when it is spoken of as the better known condition, "hour-glass contraction of the uterus".

The contraction ring is said to be caused by premature rupture of the membranes and by attempts to deliver with the forceps before full dilatation of the cervix. It is the writer's experience that cases do follow both the above causes, but that it is commonest for the condition to arise in patients in whom both the above conditions have not occurred. The condition is a serious one, and in a number of collected cases an American author gave the maternal mortality as 30% and the fœtal mortality as 50%. In fourteen cases dealt with by the writer, the maternal and fœtal mortality was 14% and 20% respectively.

Diagnosis.

The ring can sometimes be felt on abdominal examination as a depression on the outer surface of the uterus, which becomes more obvious during contraction of the uterine muscle. It can be recognized for certain by passing the hand into the uterine cavity and feeling the inner surface of the ring.

Treatment.

The most important fact in treatment is to realize that the application of traction by the midwifery forceps in the usual way will only cause the ring to become tighter. If sufficient force is used to overcome the spasm and deliver the patient, she will die of shock. Treatment is directed towards: (i) Relaxation of the spasm by drugs. Morphine and anæsthetics are of little use. Amyl nitrite by inhalation is most useful if the cervix is fully dilated and will cause sufficient relaxation of the ring to permit of delivery by forceps. Adrenaline solution (one in 1,000), 0·3 mil (five minims) given intramuscularly, is also said to cause relaxation of the spasm. (ii) Continuous weight traction. The application of the midwifery forceps is made to the head of the child, or of the cranioclast to the perforated head, and after tying the handles together firmly, traction is made by a weight of between 1·8 and 3·6 kilograms (four and eight pounds) attached over a pulley to the handles of the instrument used. (iii) Cæsarean section. Either the upper or lower segment operation will permit delivery, though the ring may have to be divided during the course of the operation. Cæsarean section should never be undertaken when the uterine cavity has possibly already become infected by such manipulation as the passage of the hand to establish the diagnosis, attempts to deliver with forceps *et cetera*.

Rigidity of the Cervix.

Rigidity of the cervix is a term applied to a condition of the cervix resulting in slow dilatation, with consequent delay in the first stage of labour. In the majority of patients said to possess a rigid cervix the term is used incorrectly, and the slow dilatation is really due to uterine inertia. True rigidity of the cervix is only found rarely, and occurs in elderly *primigravidæ* and in women whose cervix is much scarred, the result of injury in a previous labour, or after amputation of the cervix.

Treatment.

In true rigidity of the cervix the first stage of labour should be allowed to continue for several hours, to see how far dilatation will proceed under the influence of uterine contractions. When it is clear that the thin and membranous os will dilate no more, the patient should be delivered by lower segment Cæsarean section, an operation which entails the least risk to both mother and child. The patient may be said to have had a trial labour, and this should always be carried out in an institution with an operating theatre ready for use. Formerly it was usual to treat such patients by making several radiating incisions in the cervix about 18 millimetres (three-quarters of an inch) long with a pair of blunt scissors; the incidence of sepsis and the formation of still more scar tissue is to be expected from this method of treatment.

Failure of the cervix to dilate takes place when the cervix contains a fibroid or a carcinoma. These conditions, which cannot be included under "rigid cervix", should have been recognized during pregnancy and the proper treatment carried out, without allowing the patient to go into labour. They will be dealt with in a later section.

Obstructed Labour.

Obstructed labour is said to occur in patients in whom there is some mechanical obstruction and delivery cannot be accomplished by the natural forces.

Causes.

Causes fall into two groups:

1. Maternal:
   - Contracted pelvis.
   - Tumours of pelvis.
   - Rigidity of cervix.
   - Atresia of vagina.
2. Fœtal:
   - Malpresentations—
     - Transverse.
     - Brow.
     - Mento-posterior.
     - Occipito-posterior.
   - Large and post-mature infants.
   - Fœtal deformities, for example, hydrocephalus, fœtal ascites *et cetera*.

Contracted Pelvis.

Contracted pelvis is a condition in which some part of the bony canal, through which the infant must pass in labour, is smaller than normal. The two common forms are the flat type and the small, round, or generally contracted type. The ante-natal examination of pregnant women should enable most cases of contracted pelvis to be recognized before the onset of labour, and for the management of the labour to be planned in advance. Contracted pelvis should be suspected if it is found that the external pelvic measurements and the diagonal conjugate are smaller than normal. But the ease or difficulty with which the child's head will pass through the pelvis depends not only on the size of the pelvic cavity, but also on the size of the fœtal head. Therefore, satisfactory pelvic measurements are not enough to insure a safe passage of the child's head. Hence it is of greater value to compare the relative sizes of the fœtal head and the pelvic cavity than to take pelvic measurements. The importance of this cannot be over-emphasized, and in consequence the method of carrying it out will be described at length. It is done in two ways, firstly, by abdominal manipulation alone. Grasp the head between the thumb and fingers of the right hand, while firm pressure is made on the fundus with the left hand, in an attempt to push the head down into the pelvic cavity; the amount of descent is recognized by the hand on the head. If the child's head can be pushed down in this way, much more easily will the uterine muscle push it down.

A more satisfactory method is that first described by Professor Munro Kerr. It may often be of little value unless the patient is given an anæsthetic, which permits of more thorough examination by removing the pain of manipulation and the muscular resistance, which is always present, however much the patient tries to help. With the patient lying on her back, two fingers of the right hand are placed in the vagina to note the level of the head. The right thumb is allowed to project above the symphysis. The head is then pushed down by pressure upon the breech of the child by an assistant, the left hand being used to hold the head. If descent of the head occurs, it is appreciated by the fingers in the vagina. If the head of the child projects further forward than the symphysis, descent does not occur, and this greater prominence of the head is spoken of as "the head overlapping the symphysis". During labour it will be necessary for the head to mould by this amount if it is to pass through the pelvic cavity.

As working rules for the valuation of Munro Kerr's manipulation the following principles can be laid down:

1. When the head can be made to descend into the pelvic cavity the uterine muscle will push it down without difficulty, and for at least a further two or three weeks' growth of the child.

2. When the head cannot be pushed into the pelvis, but is not more prominent than the symphysis, that is, there is no overlapping, then the greater strength of the uterine muscle, together with moulding of the head, will permit of safe spontaneous delivery of the child if labour comes on within a few days or is induced at once.

3. When there is definite overlapping of the symphysis by the fœtal head, then it is more than probable that the head will be unable to enter and pass through the pelvis with safety to the child, that is, delivery *per vias naturales* is unlikely to occur and Cæsarean section will most probably have to be carried out.

Pelvic Tumours.

For practical purposes growths of this type which obstruct labour are commonly ovarian cysts. Next

in order of frequency come uterine fibroids, which arise from the cervix or lower part of the body of the uterus. Other tumours which obstruct labour are comparatively rare, but different varieties are mentioned in the text books.

Rigidity of the Cervix.

Rigidity of the cervix has been discussed in a previous paragraph.

Atresia of the Vagina.

Atresia of the vagina is a rare condition, but is occasionally met with, and is due to vaginitis occurring during the course of an acute specific fever. The patient is so ill that the pelvic condition is overlooked, and recognized only after it has produced dyspareunia, or in the course of the ante-natal examination.

Fœtal Causes of Obstructed Labour.

In the group of causes given at the beginning of this chapter it is obvious that all produce obstruction by a common factor, namely, that in all a larger diameter than normal attempts to pass through the pelvic canal. The recognition of most of the fœtal causes occurs only in delivery, and some of them, that is, brow, transverse, face presentation, may not be present before the onset of labour.

The Effect of Obstruction on Labour.

In a patient in whom no abnormality is suspected, labour commences in the usual way, the pains gradually increasing in strength. If the obstruction is at a high level and prevents the head entering the pelvis, it is probable that the membranes will rupture early and full dilatation of the cervix may never occur spontaneously. If the obstruction is low enough in the pelvis, for example, funnel-shaped pelvis, to allow the fœtal head to come well down into the pelvic cavity, the dilatation of the cervix will usually be complete before the membranes rupture.

Excessive retraction of the uterus occurs only after rupture of the membranes. Before the liquor has escaped there is no immediate danger to mother or child. After rupture of the membranes and escape of the liquor, the child is likely to be injured, possibly fatally, by pressure, and the mother is likely to become exhausted as a result of tonic contraction of the uterus.

Tonic contraction of the uterus is a condition in which the contractions follow one another with such rapidity that no interval can be detected between them; it is a spasm of the whole of the musculature of the upper uterine segment. Associated with the spasm of the upper uterine segment over-distension and thinning of the lower segment occur, and sometimes a definite ring can be felt running around the uterus at the junction of the thickened upper segment and the thinned lower segment. This is spoken of as the ring of Bandl.

Diagnosis of Obstructed Labour.

Obstructed labour can be foreseen during the systematic ante-natal examination which should be carried out on every pregnant woman in the last six weeks of pregnancy. The probability of its development in labour is recognized by: (i) determination of the presentation, (ii) comparison of the size of the fœtal head and the pelvis, (iii) discovery of other abnormalities.

If these points were investigated in every pregnant woman, obstructed labour would practically never occur. It is recognized, should it develop in labour, by the absence of advance of the child in the presence of good contractions. As labour proceeds and the pains become more frequent, the patient begins to show signs of exhaustion (general, not uterine) by quickening of the pulse rate and later by a rise of temperature. Her mouth becomes dry and vomiting is frequently seen. If the condition is allowed to continue until tonic contraction has developed, then these general symptoms are more marked, and on abdominal examination the uterus is tender, hard and never relaxes, and the ring of Bandl may be felt. While on vaginal examination, the vaginal walls are dry and swollen; the presenting part, if felt, is impacted.

The result of obstructed labour and tonic contraction, if unrelieved, is usually rupture of the uterus, beginning in the over-stretched lower segment and spreading in any direction. Often the fœtus and occasionally the placenta are expelled into the peritoneal cavity through the rupture.

An X ray photograph of the uterine contents is of the utmost value in determining the cause of obstructed labour when none of the usual causes can be discovered. Such conditions as breech with extended legs, hydrocephalus, locked or conjoined twins, cannot be diagnosed by any other means. This is particularly true if the patient is seen for the first time when in labour, as the uterine contractions render abdominal palpation more difficult and of less value in diagnosis.

Treatment.

Obstructed labour and therefore tonic contraction ought never to occur if the ante-natal examination has been efficiently carried out in all patients. But as the condition does occur and will continue to do so, its treatment will be described.

The important point is to recognize the cause of obstruction, as treatment, to be satisfactory, must depend upon the cause. Abdominal palpation will reveal nothing if tonic contraction is present; usually in these circumstances it is impossible to identify the different parts of the child. It is necessary in such a case to make a vaginal examination under full surgical anæsthesia, with the whole hand passed into the vagina. Any of the causes enumerated at the beginning of this section may be discovered. Great care must be exercised in introducing the hand, for the purpose either of diagnosis or of treatment, as the over-stretched and thinned lower uterine segment ruptures with striking ease.

Another factor which should influence treatment is that in all cases of definite tonic contraction the child is dead, and that method should be chosen, therefore, which will expose the mother to the least risk, without regard to the child. In the commonest type of case, where the head of the child is impacted above or in the pelvic brim, craniotomy is the safest method of delivery, provided the size of the pelvis is sufficient to allow the passage of a mutilated fœtus. For practical purposes delivery should be attempted after a destructive operation only if the true conjugate measures 6·8 centimetres (two and three-quarter inches) or more. The forceps should never be applied when it is known that the child is dead, and therefore never in tonic contraction. Perforation is not difficult when the head is fixed, providing the fingers guiding the perforator can reach the head. There is more danger to the mother in the extraction of the perforated head, with its extremely sharp edges of bone. Extraction of the child is better carried out by the cranioclast or the combined perforator and cranioclast, and whatever instrument is used, there is always the danger of rupturing the already attenuated lower uterine segment. When the traction is applied, it should always be made in the axis of the pelvis and a hand inserted into the vagina to be certain that the sharp edges of bone do not tear the vaginal walls.

When an impacted shoulder presentation is the cause of obstruction, decapitation is the only method of delivery, and then only if the pelvis is large enough to permit delivery, that is, true conjugate greater than 6·8 centimetres (two and three-quarter inches). Owing to the necessity of passing the decapitating hook over the child's neck there is even greater danger of rupture of the uterus than in perforation. The hook is passed into the vagina protected by the fingers of one hand and guided over the neck with its point towards the mother's back. The point of the hook must be guarded by the left hand, while the right hand pulls firmly and drags the handle of the hook up and down (the movement of a pump handle). The neck is quickly cut through, the hook removed, and the body delivered by traction on the arm; the head is afterwards perforated and delivered by the cranioclast.

When the child is alive, different methods of treatment have to be undertaken, as every medical practitioner has the great revulsion to destroying a living child in order to complete delivery. Therefore the choice will be between Cæsarean section and Cæsarean section followed by hysterectomy. Hysterectomy sounds a desperate operation for this condition, but it must be remembered that in all of them the uterus is infected, and in such cases the mortality from Cæsarean section is very high.

Where the obstruction is due to an ovarian cyst impacted in the pelvis, an attempt should be made under anæsthesia to push the cyst out of the pelvis and deliver the child. If this is successful the cyst should be removed as soon as the patient has recovered from the effects of labour, as it will have been damaged by the pressure and become infected. If the cyst cannot be pushed up, the abdomen must be opened and the cyst lifted out of the pelvis, the child should be delivered *per vias naturales* by an assistant, then the cyst removed and the abdomen closed. It is unnecessary to do a Cæsarean section when the obstruction is an ovarian cyst; the uterus is normal and will empty itself after removal of the tumour. After performing Cæsarean section an increased risk of sepsis and a weak uterine scar are both possible complications.

When a fibroid of the uterus causes obstruction, Cæsarean section is the only method of delivery; this is followed in suitable cases, which are rare, by myomectomy, but more often it is necessary to perform hysterectomy.

EMPYEMA.

By John B. Hunter, M.Ch., F.R.C.S.,
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Pus in the pleural cavity is a sequela of a previous pathological condition. That is, in the majority of cases it follows an infective process in the underlying lung. Pus in the pleural space, or empyema, at one time was treated as a surgical emergency, but following the great mortality of this practice in large influenzal epidemics, this is no longer so, and experience has shown that the indiscriminate open procedures on empyema are fatally dangerous. The present method of dealing with the condition is by a more cautious approach, allowing time for a full investigation of the character of the causative organisms and the reaction of the pleura to their presence.

The disease follows the infection of the lung at varying periods either towards the end, as in the case of lobar pneumonia, where the infection is with the pneumococcus, or while the lung infection is still spreading, when the organism is commonly the streptococcus. The recognition of these facts is of considerable importance in the prognosis and treatment of the condition.

In cases where the pneumococcus is present the patient has usually had a definite pneumonia which is resolving, the temperature settling, or settled, with general constitutional improvement; this is followed by a further rise of temperature and fresh signs indicating an effusion into the pleural cavity.

On the first suspicion that fluid is present a needle should be introduced into the pleura. If fluid is withdrawn, this is usually purulent or already definite pus; in some cases the pus is so thick that it cannot be withdrawn, and only an examination of the hollow needle reveals its presence. No attempt should be made to withdraw any large quantity at this stage, but enough should be withdrawn for examination. If the pneumococcus is present, the pus is usually thick and creamy, and the pathological changes in the pleural cavity can be assumed to be those of a local

empyema, varying in size, but with definite adhesions of the lung to the parietal pleura, so that the mediastinum is fixed. This fixation of the mediastinum is of the greatest importance in relation to treatment.

If an opening is made into the normal pleural cavity there is immediate collapse of the lung on the opened side, the degree of collapse depending on the size of the opening; but if the opening is larger than the glottis, the collapse will be complete. Not only is there collapse, but the mediastinum is also displaced to the other side. This may cause immediate death or great embarrassment to the patient with the flapping of the mediastinum. If, however, the visceral and parietal pleura are adherent from disease, the opening into the pleural cavity causes only moderate collapse of the lung and no disturbance of the mediastinum, as this is fixed by the adhesions.

It will be seen, therefore, that the opening of the pleural cavity to drain an empyema can be safely performed when adhesions have anchored part of the lung and mediastinum, but such an opening in the absence of such fixation adds greatly to the patient's risks.

Where the causative organism is the pneumococcus and the pleural effusion has occurred towards the end of the lung infection, the fixation of the mediastinum is rapid, and in the presence of thick pus there is no danger of mediastinal flapping, and an opening into the cavity to allow drainage is attendant with very little risk.

In the case of a streptococcal or mixed infection of the lung the involvement of the pleural cavity takes place at an earlier stage, while the lung infection is at its height or still progressing. Where the presence of fluid is suspected, aspiration will usually reveal a cloudy, thin fluid. However heavily infected examination shows this fluid to be, open drainage should never be undertaken at this stage, as a free pleural cavity may be encountered with the formation of a pyopneumothorax and the immediate dangers of a flapping mediastinum. Aspiration of the fluid from time to time will give the patient relief from the grosser toxic symptoms and allow time for the formation of adhesions, this latter being usually indicated by the purulent fluid turning to thick pus.

The above remarks indicate the general principles which should guide us in the treatment of pus in the pleural cavity.

The details of treatment are similar. When thick pus has been withdrawn, it may be assumed that the period when aspiration can be employed is over and that adhesions have formed. More adequate drainage must now be provided. This is done by removing a portion of rib and inserting drainage tubes. The patient is given 0·015 gramme (one-quarter of a grain) of morphine three-quarters of an hour before the operation, and local anæsthesia is employed. The patient is placed on the sound side on the table and an incision made along the eighth rib in the posterior axillary line, care being taken not to make it too far back. When the rib is reached about 7·5 to 10·0 centimetres (three to four inches) are removed and a hollow needle is inserted to make sure that the pus is underlying the pleura. Pus having been withdrawn, a longitudinal incision is made in the pleura. A finger is then introduced and large flakes of lymph are gently separated, and loculi, if present, are very carefully broken down.

Two procedures are then open to the operator, either to place a large tube just inside the cavity and close the opening tightly round it, and so produce a temporary closed drainage, which drains by means of a longer tube under the surface of an antiseptic fluid in a bottle, or to place three tubes side by side, like the "pipes of Pan", just into the cavity and bring them on the surface and allow open drainage.

This latter method is one that I advocate. It requires more attention from the surgeon, but in my experience is most satisfactory. The after-treatment differs slightly, according to whether the empyema is a pneumococcal or a mixed infection. In the former a carbolic dressing, one in forty, is applied and changed daily, in some cases twice a day. The tubes are removed on the fourth day and after that time the carbolic dressing is still continued and no attempt is made to introduce a drain into the wound. In the majority of cases healing takes place without any complications and the lung reexpands. If any doubt arises as to a recollection of fluid, sinus forceps should be introduced, but usually one's fears are not justified and in no case is it necessary to reintroduce a tube.

In the case of the mixed infection the tubes have to be left for a longer period, but rarely more than ten days, and eusol irrigation is employed from the fourth to the eighth day.

In both cases blowing exercises are beneficial, not necessarily with the idea of expanding the lung, but to make the patient use the intercostal muscles on the affected side; this is of considerable importance in reestablishing the full function of the lung.

Inadequate drainage is unquestionably a cause of chronic empyema, but in my opinion the introduction of a tube into the pleural cavity for too long a period is almost more harmful. A serous surface, such as the pleura, which has been inflamed, will continue to discharge for a considerable period, and to leave a tube *in situ* will continue the irritation and prevent the natural healing process. Chronic empyemata will frequently be met with and their cause is not always clear, although a large list of causes can be enumerated by any third-year student, and such a list suggests at once that our knowledge on the subject is very superficial. I should like to reduce them to three: (i) inadequate drainage of the empyema cavity when the rib is resected, (ii) the employment of tube drainage over too long a period, and (iii) the presence of an intermittent reinfecting cause, such as a pleuro-bronchial fistula.

The presence of a chronic empyema calls for the most resolute treatment. Its presence is a constant

source of ill health and danger to the patient, and usually makes him or her an inefficient wage-earner.

In treating such a case the extent of the chronic cavity must be investigated. This is done by introduction of lipiodol through the sinus. It is not necessary to fill the cavity completely; in some it is almost impossible; its extent can be estimated by taking three skiagrams, one with the patient in the upright position, one with the patient lying on his back, and one lateral. An accurate estimate of the size of the cavity can thus be made and the subsequent procedures can be planned.

As a general rule it can be laid down that a collapsing operation will give the best results, either done in one or more stages, the overlying ribs and greatly thickened parietal pleura being removed; it is possible to do this leaving the intercostal nerves intact. The wound produced by such an operation should not be closed, but allowed to granulate from the bottom, oiled silk being employed next to the raw surface to avoid painful dressings.

Decortication has not in my experience given the results that are claimed for it. The greatest difficulty is freeing that part of the pleura where the reflection is taking place from the parietal and visceral pleura, and in the very nature of the case this is the most important part to free and the most difficult to get at without adequate exposure, which in itself may contribute very largely to the success of the operation.

No case of chronic empyema should be allowed to drift on without an attempt being made to remedy the condition, and it should be remembered that the superficial examination of a sinus and its exploration with a probe may give very little informaton as to the underlying condition, and that X ray examination with an opaque fluid is the only means of determining the full extent of the lesion, which can in a large number of cases be cured.

THE PRACTITIONER'S CARE OF INSULIN DIABETICS IN ROUTINE AND EMERGENCIES.

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WHETHER the initial insulin treatment is undertaken in a hospital or by a specialist, the diabetic sooner or later returns home to his practitioner's care. The initial treatment consists in working out the correct dose of insulin, when necessary, to metabolize whatever diet is chosen for the patient, diet alone having failed to control the diabetes. This dosage is always an individual one and has to be varied from case to case, depending on the severity of the disease as well as the diet, particularly the amount of carbohydrate. The one essential of the treatment is that the carbohydrate is regularized from day to day and the correct dose of insulin injected to balance it and prevent both hyperglycæmia (an excess of sugar) and hypoglycæmia with its unpleasant symptoms from a temporarily subnormal blood sugar.

When this initial balance is attained, the patient —trained to look after his diet, injections and usually his urine tests—returns to his home and occupations. If all goes well, his doctor may see very little of him, sometimes for months on end. But when the routine goes wrong or the patient gets ill with some other disease, the doctor is at once consulted. He is often placed in a difficult position, as he knows, in some ways, less of the detailed management of the case than the patient himself—and this is bound to be so with intelligent patients. The doctor, however, is expected to know everything and to put matters right.

It may be useful, therefore, to outline: (i) changes that occur in the routine and the adjustments of treatment required, and (ii) difficult emergencies that may arise.

Routine Adjustments of Insulin.

It might be thought that, if the diet is kept constant, the same dose of insulin would suit the patient indefinitely. This is rarely so, and in most cases the insulin requires to be reduced or increased some time or other.

A reduced insulin requirement is shown by the frequent occurrence of hypoglycæmia with symptoms of weakness and shakiness at regular times (usually three to five hours) after the dose of insulin. This quite commonly occurs at the beginning of treatment, when the patient's own carbohydrate tolerance may progressively improve. A few, especially the elderly, are able to leave off insulin altogether as months progress. If the necessary reduction of insulin is not made, the patient feels weak and wretched.

Improvement in general health and especially activity usually reduces the insulin requirements. Exercise or manual labour always does, and it is frequently necessary to prescribe a smaller dose on days when unusual exercise is taken. I find that patients who leave hospital and return to an active life at once, need less insulin and readjustment of the hospital dose to ordinary work-day conditions.

Increased insulin requirements, shown by the reappearance of sugar in the urine and a raised blood sugar, are brought about by the converse circumstances, poor general health, fatiguing overwork, and especially any worry or mental strain. Less exercise or too great an increase in weight may also require more insulin; in the latter case the diet should be reduced at least in fat (that is, total calories). Colds and minor infections may require an increase of insulin, at least temporarily. It should also be remembered that, especially in the young, there is sometimes an inherent tendency for the diabetes to grow worse, and permanent decline in tolerance is easily caused by minor infections or by periods of carelessness in diet.

If such changes are not met by giving more insulin, the sugar gradually becomes worse and the

patient's condition deteriorates to such an extent that severe ketosis and even coma may ultimately supervene.

Diagnostic Table.[1]

	Diabetic Coma.	Insulin Coma.
Onset	Gradual	Very sudden
Tongue	Dry	Moist
Respiration	Deep; air hunger	Quiet
Pulse	Feeble, rapid	Normal or full and rapid
Temperature	Subnormal at first	Normal
Skin	Dry, blue colour	Sweating
Dehydration	Marked	Absent
Eye Tension	Usually low	Normal
Gastric	Vomiting	No vomiting
Nervous system	No pathognomonic signs	
Reflexes	Usually absent	Normal or brisk
Pupil	Contracted or normal	Often dilated
Laboratory tests—		
Urine sugar	+++	Absent or traces
Ketones	+++	Absent or traces
Blood sugar	Hyperglycæmia	Hypoglycæmia
Alkali reserve	Very low	Normal

[1] From "The Diabetic Life", Seventh Edition, J. & A. Churchill.

It should be remembered that the presence of some glycosuria does not always mean that insufficient insulin is being given. This entirely depends on the time of day the sugar is present. It is rarely possible in severe cases treated with two doses a day (and this is usually enough) to keep the urine entirely sugar-free throughout the twenty-four hours. It is usual to find that, although the urine may be sugar-free most of the night, the blood-sugar tends to rise about 7 or 8 a.m., when the action of the previous evening's insulin is quite worn off. Therefore, before and after breakfast, it may be impossible to prevent an hour or two of hyperglycæmia and glycosuria. After the morning insulin this disappears, and the same patient may be sugar-free by noon with a normal blood sugar; indeed, he may be hypoglycæmic—such rapid changes does insulin produce within a few hours. The same period of permissible glycosuria may occur round the evening meal, when the morning insulin has worn off and before the evening dose has exerted its full effect. If, however, the urine secreted when the insulin action is at its maximum (say, three to six hours after the injection) contains sugar, this shows that the diabetes is never being controlled, and that more insulin is necessary. It is obvious that in such cases a total day's collection of urine will hardly ever be sugar-free, and if the insulin is raised in the attempt to make it so, hypoglycæmia is likely to occur at some time in the day. Short-period collections of urine are much better, and I often tell such patients to test the urine passed five hours after insulin, the bladder having been previously emptied three hours after insulin. If the five-hour specimen is sugar-free, then routine control may be considered satisfactory. As an ultimate guide to the best dosage, I consider a blood sugar test about four to five hours after insulin (that is, before the midday meal) the most satisfactory, and the insulin should be adjusted to make this normal, 0·100%

Emergency Conditions.

The practitioner is faced with far more serious difficulties when sudden illness or accidents interfere with the patient's usual routine. Every febrile infective process diminishes the usual action of insulin and almost invariably makes a larger dose necessary. The upset may be so great, especially in children, that, if the necessary adjustment of treatment is neglected, severe ketosis and actual coma may be precipitated in two or three days. Poisoning (diabetic ketosis or "acidosis") from accumulating ketone or acetone bodies is the danger, and an outline of the preventive treatment necessary may be useful. The presence of gastro-intestinal disturbance (nausea, vomiting, or diarrhœa) makes treatment especially difficult, and will be considered separately.

Illness Without Gastric Upset.

Where the usual diet can be eaten, little trouble will be met. The usual total amount of carbohydrate should be given, perhaps in a lighter form, suitable for a febrile condition, and the ingestion of the usual protein and fat foods does not matter for a few days. The urine must be tested at regular times throughout the day, and if sugar appears when it is usually absent, the insulin must be increased sufficiently (perhaps even doubled) to control it. It is often wise, to obtain this control, to give three or four doses of insulin at eight-hourly or six-hourly intervals, instead of two very large doses twice a day. If the sugar is controlled in this way, one may be certain that the ketosis is negligible.

Illness With Gastric Upset.

The essential is not to stop the insulin, a mistake which patients tend to make. They may think that when food is not eaten to balance the insulin, the latter should be stopped for fear of an overdose. This idea has been productive of the greatest number of coma or precomatose conditions which I have met in the last eight years. If no food can be retained (very uncommon) half the usual dose of insulin should be given and the case treated as a grave emergency for immediate admission to hospital. But almost invariably the usual amount of carbohydrate will be retained if given in suitable form. Sugared drinks flavoured with lemon, orange juice, or, better, Benger's, Horlick's, or some similar starchy gruel, can be retained at regular intervals (two or four hourly) balanced with enough insulin to keep only a trace of sugar in the urine. If vomiting continues, the necessary carbohydrate must be given as glucose by rectum, subcutaneously, or even intravenously. Such cases are grave emergencies, and the most efficient treatment is almost impossible to carry out at home.

The Unconscious Diabetic.

When a doctor is suddenly called to an unconscious patient, known to be a diabetic on insulin, he may have a difficult and immediate decision to make if the patient's life is to be saved.

DIABETIC KETOSIS—EMERGENCY TREATMENT.[1]

Diet and Insulin Control by Urine Tests.

Diet.—Carbohydrate, *40* grms.[2] The following contain 10 grms.:

By day, ¼-hourly.
By night, ½-hourly.

Sugar, 2 large lumps or 2 teaspoons.
Glucose, ½ oz.
Benger's, 2 level teaspoons.
Horlick's, 2 heaped teaspoons.
Ovaltine, 2 very full teaspoons.
Orange juice, 4 oz.
Milk, 7 oz.
Give water between feeds.

Insulin.—Test urine for sugar before each dose.

Give	At	Units
½-hourly	6 p.m.	*40* if Benedict's test is red or yellow
	10 p.m.	= +++ sugar.
	2 a.m.	*30* if green with copper deposit
	et cetera	= trace.
		20 if blue = no sugar.

Date.	Time.	Urine Sugar.	Ketones.		Insulin.	Carbohydrates.
			FeCl.	Roth.		
21.3.32	6 p.m.	+++	+++	+++	40	40 grms. (40 grms. glucose + lemon juice)
	10 p.m.	+++	+++	+++	40	40 grms. (40 grms. glucose + lemon juice)
22.3.32	2 a.m.	+++	++	+++	40	40 grms. (8 teaspoons Benger's)
	6 a.m.	+++	0	+++	40	40 grms. (8 teaspoons Benger's)
	10 a.m.	Trace	0	++	30	40 grms. (8 teaspoons Benger's)
	2 p.m.	0	0	+	20	40 grms. (8 teaspoons Benger's)
	6 p.m.	Trace		0	30	40 grms. (8 teaspoons Benger's)
	10 p.m.	Trace		0	30	40 grms. (8 teaspoons Benger's)
23.3.32	2 a.m.	0		0	0	0
	6 a.m.	+		0	30	40 grms. (8 teaspoons Benger's)
		Put on 120 grms. C. + insulin 40 b.d.				

[1] From "The Diabetic Life", Seventh Edition, J. & A. Churchill.

[2] The figures in italics vary with the requirements of each case.

The patient may be suffering from an overdose of insulin on the one hand, or, on the other, from true diabetic coma, produced by omission of insulin or an inadequate dosage. Treatment is urgently required; sugar or glucose to relieve hypoglycæmia, or more insulin to treat diabetic coma. The problem is one which will very seldom arise in any one doctor's practice, and the accompanying table, which summarizes the diagnostic points, may be helpful.

It will be seen that on clinical grounds alone there is little difficulty in making the diagnosis. If the onset is abrupt, this almost rules out any suspicion of true diabetic coma and in the absence of cerebral hæmorrhage or some such complication makes an overdose of insulin almost certain. The distinction between the dry parched tongue of diabetic coma and the normally moist tongue in an overdose of insulin is also invaluable and readily observed.

The treatment of insulin coma consists in giving soluble carbohydrate in sufficient quantity to raise the blood sugar to normal. Sterilized glucose given subcutaneously, or, better, intravenously, acts most quickly, and by the latter route may produce a complete and dramatic recovery in one or two minutes. Where sterile glucose solutions are not available, a sugared drink should be introduced into the stomach, by sips by mouth or by nasal catheter or stomach tube. An ounce (30 grammes) of sugar is ample to restore the patient, though more may be required later in unusual cases. On the other hand, for diabetic coma, frequently repeated large doses of some fifty units of insulin may be necessary for many hours.

If any doubt exists about the diagnosis in the unconscious diabetic, sugar should be given at once. An ounce will rapidly restore the patient with insulin coma and by so doing quickly confirm the diagnosis. At the same time this sugar will do no real harm to the patient with diabetic coma where insulin can be given as soon as possible after the diagnosis is certain. There is no real difficulty in differentiating the two conditions when it is possible to bring a considered judgement and little knowledge to bear on the circumstances. But the emergency of a sudden night call and scanty data to work on may create real difficulties in which it is well to remember the rule to give sugar at once and decide a little later.

The Treatment of Precomatose Conditions.

This is no place to give the full details of treatment of diabetic coma and, when fully established, it is best treated by a large initial dose of insulin (say, fifty units) and immediate transfer to a hospital or home where the elaborate and prolonged treatment often necessary can best be carried out. But if the case is less severe and the patient can still swallow and retain feeds by mouth, the following chart outlines an easy and efficient method of treatment.

The essential is to give regular amounts of carbohydrate by mouth at set intervals and regular large doses of insulin. The dosage should be adjusted by urine tests, as shown in the table, and in this way there is no fear of causing an overdose. With this energetic sugar and insulin treatment and abundance of fluids all precomatose cases should be saved.

THE DIAGNOSIS AND TREATMENT OF HOARSENESS.

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In choosing a subject which will be of interest to the general practitioner and shall at the same time give him some assistance in dealing with cases, it has seemed advisable to the writer to take the above title. Hoarseness is a symptom frequently met with; it may be of little significance, but, on the other hand, it may be of the most manifest importance to discover the cause. If the latter be not brought to light and dealt with, the patient's life may, in a considerable number of instances, be endangered. The subject is a large one and comprises the greater number of diseases of the larynx, all of which upset the voice.

Definition.

By "hoarseness" is meant some upset of the speaking voice through which a rough character is introduced. Before considering the mechanism by which this is brought about, it is advisable to say a few words on the subject of phonation in man.

Mechanism of Phonation.

During voiced speech the vocal cords are brought together by contraction of the various constituents of the sphincteric muscle girdle. The muscles particularly concerned in this mechanism are the thyreo-arytenoid, lateral crico-arytenoid and inter-arytenoid, all of which, by synergic contraction, approximate the arytenoid cartilages and vocal cords. If they acted without opposition, the arytenoids would be pulled forwards towards the thyreoid cartilage; this would be undesirable from the vocal point of view. Therefore the arytenoids are braced back by the posterior crico-arytenoid muscles and the vocal cords are kept extended.

During phonation the vocal cords are closed and an expiratory effort is made by which the pressure of air in the trachea is raised. By virtue of the contraction of the thyreo-arytenoid muscles a certain degree of elasticity is given to the vocal cords. This elasticity varies with the strength of contraction of the thyreo-arytenoid muscle. During a high pitch the thyreo-arytenoid contracts more powerfully than during a low one. As the pressure in the trachea rises it blows apart the membranous margins of the glottis, namely, the vocal cords. The arytenoid cartilages should in correct methods of phonation remain in firm apposition. A puff of air escapes through the glottis and thereby the air pressure in the trachea is momentarily lowered; the elasticity of the glottic margins causes the vocal cords to come into apposition during this lowering of pressure, but immediately they do so the pressure again rises and the cords are blown apart. If this escape of puffs of air takes place in a rhythmical manner, a musical sound is produced. The pitch of the sound depends on the rapidity with which puffs escape; this in itself depends upon the degree of elasticity of the glottic margins and the air pressure in the trachea.

Pathological Changes Causing Hoarseness.

Weakness of Laryngeal Muscles.

If the glottis be not efficiently closed, as just described, the margins will not come together firmly between the escape of each puff of air, and some air will be allowed to blow through at these times. This will give a "breathy" character to the voice.

The reason for lack of approximation may be either functional or organic. Many cases are met with in which the patients do not bring the vocal cords together, and air waste occurs, with feebleness of the voice and often with some degree of hoarseness. The sound then approximates to a whisper. In these cases there is no actual paralysis of the cords, as may be discovered when the patient coughs. In this action the vocal cords come close together. The lack of approximation may be entirely functional and of an hysterical nature, but in some cases, as in pulmonary tuberculosis, there is actual weakness of the laryngeal muscles, which makes it impossible for the vocal cords to be held firmly together.

In organic paralysis there is real inability to close the glottis. One vocal cord is paralysed because of some interference with its nerve supply, namely, the recurrent laryngeal. Not only is it prevented from meeting its fellow firmly, but also it is deprived of the firm support of the thyreo-arytenoid muscle, which gives it elasticity; it therefore blows open during phonation more readily than its firmly supported fellow and allows air to escape at a different rate, whereby dysphonia is produced with a hoarse character to the voice. In other cases the paralysis may be due to bulbar palsy caused by lesions in the medulla. In this disease other changes take place in the muscles of the pharynx and tongue, to be referred to later.

Paralysis of the vocal cords is occasionally met with in other organic nervous diseases, notably syringomyelia, tabes and general paralysis of the insane.

Excessive Contraction of Laryngeal Muscles.

In some hysterical subjects the ventricular bands are brought together, in addition to the vocal cords, in the condition known as hyperphonia; air is prevented thereby from escaping freely through the glottis, and a husky character is given to the voice. The same condition is found after some operations on the larynx, notably after removal of one vocal cord. Difficulty in closing the glottis, because of the absence of one cord, causes the patient to bring his ventricular bands together, with the production of hoarseness of a character rather different from that usually associated with the results of the operation.

Roughness of Glottic Margins.

If a pure sound is to be produced, the laryngeal margins must be sharp-edged, so that they shall

come together along the whole length at precisely the same moment. If one part closes before another, air escapes at irregular intervals and gives a rough character to the voice. Consequently it is found that if there is inflammation of the vocal cords, hoarseness is produced. This may happen in acute or chronic laryngitis of simple nature. It will be found more noticeably in cases of tuberculous laryngitis in which there is infiltration of the glottic margins, and sometimes ulceration.

Mechanical Obstruction to Movements of Vocal Cords.

There may be no actual weakness of the intrinsic muscles of the larynx, but interference with the movements of the arytenoid joints on one or both sides, by inflammation; this occurs in some cases of tuberculosis or perichondritis. In other instances there may be infiltration in the neighbourhood of the joints, preventing movement. This occurs in cases of malignant growth.

Mechanical Prevention of Approximation of Glottic Margins.

If something comes between the vocal cords they will not be able to meet and a gap will be left through which air escapes, leading to a fricative or "breathy" sound, causing hoarseness. There may be deposits on the vocal cords, such as take place in diphtheria or in atrophic laryngitis, when crusts form. In both these cases the obstruction is due to inflammatory exudate.

In people with high-pitched voices, as for instance, sopranos, there may be little hæmorrhages at the junction of the anterior and middle thirds of the cord, with the formation of a thickening due to heaping up of squamous epithelium. These thickenings are known as "singers' nodes". In individuals with deep voices proliferation of the epithelium may take place over the tips of the vocal processes of the arytenoids. Wart-like excrescences form on the vocal cords and prevent the approximation of the two margins of the glottis. This condition is known as *pachydermia laryngis*.

The conditions so far described are inflammatory, but the obstruction to approximation is in many cases caused by formation of a new growth. The only common innocent new growth is papilloma, which grows at the anterior end of one or both vocal cords. This is found almost invariably in children. In elderly men the only common type of growth is carcinoma. This grows at the anterior end of one vocal cord and prevents efficient closure of the glottis.

Interference with Resonation in the Pharynx.

Vocal sounds are produced by the larynx, but they are modified materially by resonation in the pharynx and mouth. If the movements of the pharyngeal muscles be prevented, as by acute or chronic pharyngitis, a change in the voice, of a husky character, may be caused. Similarly, if the movements are prevented and the cavity of the pharynx is partly occupied by a new growth, the voice is altered.

Incidence.

Hoarseness is met with at all ages, from small infants to very old men. By noting the age of the patient very considerable assistance can be gained in diagnosis. Therefore some consideration must now be given to the diseases affecting individuals at different ages.

Children.—In children simple laryngitis may occur in association with pharyngitis and rhinitis. This can happen during an ordinary cold or during an attack of an infectious disease, such as measles or scarlet fever. Inflammation of a specific nature, causing hoarseness, occurs in the form of diphtheria. Tuberculosis can affect children as young as ten, but is extremely rare and can therefore be left out of consideration. Paralysis of the larynx does not affect children, nor do such complaints as singers' nodes or pachydermia; the field is thereby very much limited. Malignant disease is left entirely out of account, as it is a complaint of elderly men. Papilloma, on the other hand, is a disease of children, commencing usually at the age of four or five and disappearing after a few years. It never leads to malignant changes.

Young Adults.—Young adults do not suffer from papilloma nor from carcinoma. They may have acute or chronic laryngitis, with the associated symptoms in the air passages above the larynx, that is to say, the nose and pharynx. Tuberculosis of the larynx is particularly a disease affecting young adults between the ages of twenty and thirty. Men and women suffer equally. Syphilis can affect the larynx, but it is so extremely rare that it is possible to leave it out of account for all practical purposes.

Middle-Aged Women.—Functional aphonia is a fairly common complaint of youngish or middle-aged women of a neurotic nature. They may suffer from simple inflammations or from one or other form of organic paralysis of the larynx. They will not be affected by papilloma, except in very rare instances, and practically never by intrinsic carcinoma. They are the subjects, however, of carcinomatous growth, commencing behind the cricoid cartilage and spreading upwards into the pharynx, thereby producing a change in the voice.

Middle-Aged Men.—Here again the simple inflammatory diseases are met with, and the various forms of organic paralysis. Carcinoma seldom occurs below the age of forty-five. Papilloma is, as already stated, a disease of childhood, tuberculosis one of adults, and singers' nodes one of sopranos, so that the field is very much narrowed.

Elderly Men.—Hoarseness commencing in an elderly man points immediately to a diagnosis of carcinoma of intrinsic type. If the disease is discovered in early stages, it can usually be cured; therefore the suspicion must always be held that a malignant growth is present, and nothing must stand in the way of the establishment of a diagnosis. If the history is given that the hoarseness has only recently appeared, it is highly improbable that it is due to chronic laryngitis of a simple nature,

because the latter is a complaint that will have affected the individual rather earlier in life. It may, of course, have recurred from time to time, but will not appear for the first time in his old age.

Senile tuberculosis is met with, but only exceptionally. Pachydermia affects elderly men, but its onset is spread over a space of some years. It occurs in those who use their voices overmuch, notably in newspaper sellers and street hawkers, while carcinoma does not appear to bear any relation to the use of the voice.

Diagnosis.

By the Voice.

The voice is feeble and "breathy" or even absent in functional aphonia; similarly in tuberculosis. In acute laryngitis phonation is painful and the pitch is lowered; in chronic laryngitis it is rough, without pain.

Of new growths, papillomata gives a husky character associated with stridor. An elderly man with intrinsic carcinoma has a rough and often powerful voice without pain on speaking.

A pharyngeal growth gives a voice sounding as if a lump or plum were in the throat.

Central nervous lesions, such as bulbar palsy, have associated difficulties of articulation.

Diagnosis by Associated Symptoms and Signs.

Pain.—If pain is present, the condition may be due to acute laryngitis or to tuberculous laryngitis with ulceration. The pain is then felt at the level of the larynx. In extrinsic carcinoma of the larynx or in carcinoma of the pharyngeal walls there is characteristic pain radiating up to the ear.

Dysphagia.—Dysphagia is associated with pharyngeal carcinoma, and particularly with the post-cricoid carcinoma which occurs in women. In the latter case the hoarseness of voice is caused by swelling of the aryepiglottic folds from upward extension of the growth. This swelling in turn impedes the movement of the arytenoid cartilages. Dysphagia is present in cases of bulbar palsy, which is, however, rare. It is then unassociated with pain. In advanced tuberculous cases causing hoarseness there is dysphagia, but in this case it is associated with pain, often of an agonizing nature.

Dyspnœa.—Dyspnœa in a small child may indicate diphtheria, in which case fever and other signs are present. As a chronic condition in children it is most often associated with the presence of papillomata. In young adults it is rarely present, but in slightly older people it may be due to obstruction of the glottis by tuberculomata in the lupoid type of tuberculosis. It is possible that it will then be severe enough to necessitate tracheotomy. The great majority of cases of hoarseness associated with dyspnœa are met with in elderly men who have a carcinoma in the interior of the larynx affecting one or both vocal cords. There may be, on the other hand, a growth near the aperture of the larynx on the epiglottic or aryepiglottic folds, which obstructs the airway. In this case it is very probable that pain in addition will be present.

Cough.—Cough is present in acute and chronic laryngitis of a simple nature and often in tuberculous cases. It is present also in diphtheria. It is seldom found in carcinoma.

Wasting.—Wasting associated with hoarseness almost invariably indicates tuberculous laryngitis; signs will be found in the lungs on physical or X ray examination. It is also found in very advanced cases of malignant growth, but only at a late stage, when the other signs, such as dyspnœa or dysphagia and pain, will make the diagnosis easy.

Fever.—Fever is present in simple acute laryngitis often to a high degree, particularly in those cases of œdematous or suppurative nature. It is also found in diphtheria and in tuberculosis; in the latter it may appear only in the evenings.

Inflammation of the Upper Air Passages.—Inflammation of the upper air passages is present as a cause in most cases of simple laryngitis, either acute or chronic. In atrophic laryngitis the cause is found in atrophic rhinitis. The pharynx as well as the nose is affected in most cases. Infection of the sinuses, teeth or tonsils may produce laryngitis and even mouth breathing alone, as for instance, deviation of the nasal septum, may act as the cause.

Diagnosis by Laryngeal Appearances.

Inflammatory Diseases.—Simple laryngitis, acute or chronic, causes diffuse, bilateral, symmetrical redness of the vocal cords alone or of the whole of the larynx. In atrophic cases crusts are present and the mucous membrane is glazed.

Singers' nodes appear as nodules on the vocal cords at the junction of the anterior and middle thirds; *pachydermia laryngis* is recognized by horny thickening posteriorly on the vocal cords, in the region of the vocal processes of the arytenoid cartilages.

Diphtheria produces sloughs and ulceration with fever. In tuberculous laryngitis there is inflammation of the posterior region of the larynx affecting the vocal cords and inter-arytenoid space; the distribution is usually bilateral, but always asymmetrical, thus distinguishing the condition from simple laryngitis. There is often superficial ulceration of the cords of "mouse-nibbled" type, and sometimes, in severe cases, marked œdematous swelling of the aryepiglottic folds with large ulcers, and even with perichondritis, causing external swelling. In lupus of the larynx the epiglottis and aryepiglottic folds are affected most, there being jelly-like nodules with ulceration and subsequent healing with scars; the appearance of syphilitic inflammation of the larynx—a rare disease—is similar.

New Growths.—Papillomata in children are pedunculated, nodular, red outgrowths; direct laryngoscopy is required for their examination. An intrinsic carcinoma usually involves the anterior end of one vocal cord; it is a nodular growth with patches of leucoplakia. It may spread to the

opposite cord or to the ventricular band, or to the posterior margin of the glottis. Extensive growths appear in the epiglottis or aryepiglottic folds and may spread to the pharyngeal walls; they are reddish, nodular and vascular, and ulcerate early.

Neuroses.—In functional aphonia the vocal cords do not come together in phonation; they close, however, on involuntary coughing and open widely during respiration. If one cord is paralysed in affections of the recurrent laryngeal nerve, it appears shorter than its fellow and does not move away from the mid-line on inspiration; it may move inwards during phonation, unless the paralysis is complete.

In double abductor paralysis the cords lie close together and the resulting obstruction to respiration produces stridor and great distress. In complete double paralysis the cords lie motionless and are slightly separated, allowing some air to pass between them.

Treatment.

Inflammatory Diseases.

Simple Inflammation.—Simple acute laryngitis is associated with acute rhinitis and pharyngitis. The treatment resolves itself into the treatment of the conditions in the nose and pharynx; for the larynx itself steam inhalations containing menthol, 0·18 gramme (three grains), compound tincture of benzoin, 3·5 grammes to half a litre (one drachm to a pint) of boiling water, breathed in from a quart-sized jug every two hours or less often give considerable relief. Direct applications to the larynx are not necessary.

Simple Chronic Laryngitis.—Simple chronic laryngitis is almost invariably secondary to a cause in the nose or pharynx. The treatment therefore resolves itself into elimination of this cause either by removing nasal obstruction, as by resection of the septum, or taking out polypi, or by the cure of simple or hypertrophic rhinitis or sinusitis. Infective tonsils may need removal, and carious teeth must be attended to. For the larynx itself steam inhalations are useful, together with spraying from a nebulizer containing an oily solution, such as menthol 0·5%, *olei eucalypti* 1%, *olei pini pumilio* 1% in spray paraffin. The nose and throat should be sprayed with this solution, the breath being drawn in during the application.

Atrophic Laryngitis.—Atrophic laryngitis is caused by atrophic rhinitis and pharyngitis, which must be cured. If crusts stick on the vocal cords, they may need removal by a swab on a long, curved holder, and occasionally by direct laryngoscopy. Potassium iodide given by the mouth may be of help.

Singers' Nodes.—Singers' nodes are due to incorrect methods of voice production. The individual must be reeducated. Very occasionally it is necessary to remove the nodes by direct or indirect laryngoscopy.

Pachydermia Laryngis.—*Pachydermia laryngis* is caused by excessive use of the voice, and therefore vocal rest is required. Potassium iodide may be given; and in cases with much obstruction diathermy may be applied to the thickenings.

Inflammations due to Specific Organisms.

Tuberculous Laryngitis.—Tuberculous laryngitis necessitates rest of the vocal cords by silence or whispering and by abstention from any exercise which necessitates powerful use of the arms. Such exercise causes the vocal cords to be closed for fixation of the thorax. The patient should be treated in a sanatorium if possible.

In resistant cases, when the chest condition is quiescent, the direct cautery may be applied to the larynx, but never if the chest is active, as the general disease may flare up. Very rarely the glottis becomes obstructed and tracheotomy is required. If there is much pain on swallowing, insufflation of orthoform and anæsthesin in equal parts, used in Thomson's type of Leduc's autoinsufflator, is most useful. The patient puts the glass tube between the lips and takes a deep inspiration through the nose; the powder then shoots down on to the inflamed surfaces of the larynx.

Syphilitic Laryngitis.—Syphilitic laryngitis requires injections of arsenic and the giving of mercury and iodides.

Diphtheritic Laryngitis.—Diphtheritic laryngitis is treated by early injection of antitoxin and frequently calls for tracheotomy. In some hospitals it is now the habit to use a direct laryngoscope, by means of which the larynx is exposed and crusts are picked out with forceps.

New Growths.

Simple New Growths.—Simple new growths generally appear as papillomata in children. If there is much dyspnœa tracheotomy is required. The new growths should be picked off the vocal cords with cupped forceps used through a direct laryngoscope, great care being taken not to bite away the mucous membrane of the larynx.

Other simple new growths, such as fibroma, angioma and simple cysts, are only rarely met with and are removed by indirect, or preferably by direct, laryngoscopy.

Malignant New Growths.—Malignant new growths are almost invariably of carcinomatous type. If the anterior end of one vocal cord is affected, laryngo-fissure or thyreotomy should be performed. The ala of the thyreoid cartilage on the affected side is removed after opening the larynx and the growth is taken away with a surrounding area of healthy tissue. The results are excellent and the rule is to obtain a cure. If the growth is more extensive and if it is of an active type, as shown by Broder's classification, it will sometimes be necessary either to perform complete laryngectomy or else to apply radium. If the latter course is decided upon, the greater part of the thyreoid ala on the affected side is removed and a palisade of six or seven long radium needles, each containing 1·5 or 2·0 milligrammes of radium

element, are put in position for a week. The results are frequently very gratifying, but if a growth is obviously suitable for removal by laryngo-fissure, that course should be adopted. When both vocal cords are affected, total laryngectomy is required, sometimes with preliminary tracheotomy.

Extrinsic carcinoma on the aryepiglottic folds or on the edge of the epiglottis is treated by Trotter's method of lateral pharyngotomy. The growth is excised after removal of the thyreoid ala. If the neoplasm has involved much of the pharyngeal wall, it may be necessary to treat it by insertion of radium; the course recommended is to clear the side of the neck by block dissection of glands and to insert about sixteen or twenty radon seeds, each containing one millicurie screened by 0·3 millimetre of gold.

Deep X ray treatment has been used for these cases, particularly in France, with considerable success; but many surgeons, including the writer, prefer to remove the growth in all those considerable number of cases in which a good result may be looked for.

Neuroses.

Functional aphonia is treated by the administration of tonics and by the application of an electric current to the neck. The current need not be very strong, as the good result of restoring the voice is obtained mainly by suggestion. If the patient is closing the glottis too tightly during phonation, reeducation of the voice is required by someone skilled in this type of work, together with some mild sedative.

Organic paralysis due to interference with the recurrent laryngeal nerve can seldom be cured, as the damage is done before the patient is seen. This applies to those instances where the recurrent nerve is pressed on by an aneurysm, by mediastinal new growth or is involved in carcinoma of the œsophagus. Occasionally peripheral neuritis of the nerve is met with, sometimes of syphilitic nature, in which case treatment by mercury, iodides and arsenic may restore function. When both recurrent nerves have been paralysed, anastomosis to the phrenic may be carried out according to the method advocated by Ballance and Colledge. Restoration of function has been obtained. In other organic lesions of the nerves the treatment is that of the general condition.

CHRONIC INTESTINAL INDIGESTION IN CHILDHOOD.

By Wilfrid Sheldon, M.D., F.R.C.P.,
Physician to the Children's Department, King's College Hospital, London.

Chronic intestinal indigestion is such a commonplace among the ailments of childhood, and so often masquerades under symptoms that seem unrelated to the alimentary tract, that some consideration of the condition may not be out of place. In American literature the term chronic intestinal indigestion is often used synonymously with cœliac disease; in this country, however, cœliac disease is confined to a definite but uncommon syndrome resulting from the inability of the intestine to absorb the products of fat-digestion, which leads to great wasting, abdominal distension, and the passage of loose, large, offensive, putty-coloured motions. Cœliac disease also shows periods in which the symptoms wax and wane, but the whole course may last as long as three or four years or even longer before an ultimate recovery is made, and during the course of the disease growth ceases, so that considerable stunting remains for long after the other symptoms have abated.

Chronic intestinal indigestion is very much more common than cœliac disease, and in the majority of cases is characterized by diminished or perverted appetite, failure to gain weight, a great variety of nervous disturbances, and the passage of unhealthy, offensive motions, which often contain an excess of mucus. It was this last feature of the stools which led Eustace Smith to call the condition "mucous disease", a title which has now fallen into disuse. While in many cases the entire alimentary tract is affected, in others the brunt of the disturbance may fall on some particular portion of the digestive apparatus. Occasionally the symptoms are mainly gastric; the child frequently eructates, or may vomit a great deal of mucus, the appetite is lost, and there may be epigastric pain or, rather, a feeling of fullness and discomfort. Examination may show a large atonic stomach. In other cases the small intestine is chiefly at fault, food is imperfectly digested and hurried through to the colon, the stools are loose, brown or greenish in colour, very offensive, and contain masses of undigested food visible to the naked eye. Or the colon may be principally affected, in which case the stools may be constipated, but are more often characterized by an excessive amount of mucus. The term "catarrhal colitis" is seldom employed in the nomenclature of children's ailments, but it is applicable to these cases, which are really no more than examples of chronic intestinal indigestion. In this last group of cases the frequency of threadworm infection is worthy of mention, because it is necessary when treating this common anthelmintic infection to remember that success is likely only if as much attention is paid to improving the health of the bowel as to eradicating the worms.

Although the symptoms of chronic intestinal indigestion are most numerous at about the time of the second dentition, between the sixth and ninth years, the condition not infrequently makes its first appearance within a few months of weaning from a purely milk diet. At this age the scope of the diet is enlarging and fresh foods are being tried, but it is easy to overtax the young child's digestion, and an excess of bulky and starchy foods, such as cereals, vegetables, and raw fruits, often starts the condition. It must also be remembered that if a child is already being fed almost to the limit of his digestive capacity, any debilitating illness may

lessen the tolerance of his digestive apparatus and be the beginning of prolonged ill health. Many years ago Eustace Smith pointed out the particularly bad influence of whooping cough in this respect, and measles is almost as bad.

Causation.

There are three chief factors which lead to chronic intestinal indigestion.

Unsuitable Food.—Generally the error here lies in giving too much fermentable starchy food—potato, bread and pastry are common offenders in this respect. The surfeit of starch undergoes bacterial fermentation in the small bowel, leading to gaseous distension and colic. The contents of the small bowel are prematurely urged into the colon and thence evacuated as greenish-brown, offensive, undigested motions.

Another source of trouble are the foods which leave a good deal of residue after digestion. These foods are generally rich in cellulose, and include the root vegetables, such as carrots and turnips; peas, beans, and raw fruit, particularly when the skin is eaten; nuts, coarse oatmeal and wholemeal breads. The residue from this type of diet is bulky and irritating to the colon, which reacts by secreting large amounts of mucus. Peristaltic movements then become less effective, and constipation may follow, while the stools are large, hard, and contain masses of "white jelly" or mucus.

Mismanagement.—Many of the troubles of chronic intestinal indigestion are connected with incorrect training and mismanagement in the home, and a careful inquiry into the child's daily routine should always be made. Parents often worry far too much about their child's appetite—particularly in the case of an only child—and resort to coaxing and bribery to make the child eat up his food. Children are not slow to take advantage of this, finding that it well repays them to be difficult and finicky at meal times. Irregular feeding between meal times is often the result, appetite becomes capricious, and a taste for indigestible articles of food is likely to develop. Mismanagement in the home often first appears when schooling begins. The rush in the morning to get the child off to school is likely to mean a too hurried breakfast, with scarcely time for evacuation of the bowels. Other children whose school is too far away for them to return home at mid-day, have to make an unsatisfactory luncheon of sandwiches and are then likely to be given a heavy meal at home in the evening, which almost always leads to digestive disturbances. Eating between meals, particularly of sweets and biscuits, impairs the appetite and constitutes another important factor. Then, too, I have no doubt that in some children the stress of working for school examinations, scholarships and prizes militates against good digestion.

Chronic Naso-Pharyngeal Infection.—Chronic naso-pharyngeal infection is a frequently associated condition. Chronically infected tonsils and adenoids, or a constant muco-purulent catarrh of the pharynx is likely to lead directly to a chronic gastro-intestinal catarrh with the symptoms of a disordered digestion. A thorough examination of the throat in a good light should always be made, since treatment confined to the lower digestive tract is not likely to be successful if an unhealthy state of the throat is allowed to persist.

Early and extensive caries of the first dentition is at times a factor of considerable importance. Unless young children are able to masticate their food properly, they bolt their food in lumps, giving rise to colic and a poor digestion. Periodic examination of the teeth of young children is of great importance if coupled with treatment carried out as soon as caries begins to appear. One sometimes sees children in whom caries has been allowed to continue to such a pitch that a wholesale extraction of most of the first dentition has been deemed necessary. The benefit to health by losing so many decayed teeth is often more than counterbalanced by the interference with digestion which loss of the masticatory apparatus entails. Many of these unfortunate edentulous children, unable to take solid food, have to be fed on broths and soft cereal foods, which makes it impossible for them to grow adequately or to find sufficient energy to carry them through the programme of an ordinary child's day.

Symptoms.

The onset of chronic indigestion is gradual, and the child is likely to be brought for advice because he is not "getting on" or because he is irritable, easily exhausted, has turns in which he goes "deathly white", is puffy under the eyes, or sleeps badly.

The appetite is as a rule bad, and the child "doesn't eat a thing". Occasionally the appetite is enormous, but the food is so badly digested and poorly absorbed that it fails to make the child thrive. Often there is a craving for just those articles which are likely to aggravate the condition, such as sweets, raw fruits and raw vegetables; severe perversion of the appetite in which such odd things as coal, paper, earth and so on are relished—the condition of dirt-eating or pica—is at times a feature.

The bowels are as a rule constipated. The stools are brown or greenish, offensive, often large and at times loose, and may contain lumps of undigested food. An excess of mucus in the stool has already been commented upon. Threadworm infection is a common coincidental trouble, because the unhealthy state of the bowel forms a suitable nidus for the growth of the worms. Abdominal pains are frequent, but seldom severe. They are generally localized round the umbilicus and tend to be worse towards the end of the day. Vomiting of food is not a prominent symptom, but the child often retches in the early morning and is likely to eject mucus from the stomach where it has collected overnight.

Apart from the gastro-intestinal tract there may be symptoms referable to almost every other

system. Instability of vascular tone is indicated by sudden attacks of pallor, and sometimes fainting. The extremities are cold and often of a raw red colour, or with a bluish tinge. In many cases the temperature rises in the evening by one or two degrees and may remain slightly raised above the normal throughout the day; and this may continue for weeks or months. This low fever often passes unnoticed, which is perhaps as well, because it is likely to cause constant anxiety to the parents, who find it difficult to believe that a chronic digestive disorder is sufficient reason for the temperature remaining up.

Vague pains in the limbs are a common complaint. They tend to occur towards the end of the day, are definitely related to fatigue, and disappear when the child rests; their distribution is in the legs rather than the arms, and they are not so obviously related to atmospheric changes as are rheumatic pains. A morning cough is not unusual and is often relieved after the child has retched up mucus from the stomach.

Nervous symptoms are very common. Rapid fatigue, irritability, and lassitude are usual. Headache is often complained of and may be present at any time of day. The most troublesome nervous manifestations, however, are those which affect sleep. Often the child lies awake for hours, and when sleep eventually comes it is fitful and broken by tossing and twitching. Sleep walking and night terrors are likely, and these children are prone to attacks of croup during the night—*laryngitis stridulosa*. Nocturnal enuresis is also a common trouble.

Physical examination shows the child to be below average weight. Loss of postural tone may be evident by the child's stance, with its sagging abdomen, exaggerated lumbar lordosis, prominent scapulæ, drooping shoulders and flat chest. There may be orthostatic albuminuria. The complexion is sallow, there may be dark rings round the eyes, and the lower eyelids may be so puffy as at first to suggest nephritis. Occasionally the backs of the hands and dorsa of the feet may be puffy. The skin may be dry or greasy, and in long-standing cases the hair becomes thin, dry and brittle. Skin lesions of an urticarial nature are often present.

The tongue is as a rule covered with a thin brown fur or may appear glazed. At times the covering of fur is patchy, a red glazed surface showing between the patches of fur. Because of its resemblance to a map, this type of tongue is often called "geographical". The teeth are often carious and may be considerably ground down, particularly the incisors. The abdomen is soft, flabby and distended, but not tender. The liver can generally be felt about two fingers' breadth below the costal margin, and this should be carefully noted, because it affords one small point of distinction from cœliac disease, in which the liver is likely to be small and often cannot be palpated.

Treatment.

Although the ultimate prognosis is good, the parents should be informed that several months of diligent treatment are often required before any appreciable improvement is manifest. The two essentials in treatment are the general management of the child and the selection of a proper diet. Drugs are helpful, but are only of secondary importance.

General Management.

As a rule rest in bed is not necessary, but in the more severe cases with much abdominal distension and loss of weight preliminary treatment in bed for three or four weeks may be advisable. Otherwise the child should get up after breakfast and should be out in the open air as much as possible. A rest at mid-day is important, but it must be a complete rest, either on a couch or actually lying down on the bed. This should begin half an hour before the mid-day meal and should continue for an hour afterwards. Again at teatime the child should rest for a quarter of an hour before tea and for half an hour after. Bedtime should be early—a child of six or seven years with this condition should be in bed by half past six. Obviously under these circumstances the child cannot attend an ordinary school, at any rate during the first month or so of treatment. During the day a regular amount of exercise is necessary, but it should not be violent nor tire the child, and it is better for the child to be out playing under the supervision of an adult. Each morning before the breakfast meal the child should have a warm bath followed by a cool sponge, which, as the child gets used to it, should be changed to a cold douche, and this should be followed by a vigorous rub down with a rough towel. This improves the general tone, just as does a course of general massage, but has the advantage that it can be easily carried out at home and does not require any expert training.

Meals must be taken at regular times and should be not at all hurried, while feeding between meals must be absolutely vetoed. As a rule it is better for the meal to be taken "dry", drink being given after the meal is over. Late meals just before bedtime must be particularly avoided.

Diet.

The points to watch in the diet are that starchy foods should be at a minimum, and foods that are likely to leave a bulky residue in the bowel should be avoided. Some of the dietetic points are set out in Table I.

Drugs.

Speaking generally, the most useful drugs for this condition are mixtures of alkalis with a bitter tonic, and they should be taken before each meal. A useful prescription is: Bicarbonate of soda 0·3 gramme (five grains), tincture of rhubarb 1·2 mils (20 minims), tincture of *nux vomica* 0·12 mil (two minims), spirits of chloroform 0·12 mil (two minims), and compound infusion of gentian to eight

Table I.

Foods Not To Be Taken.	*Foods That May Be Taken.*
Coarse oatmeal.	Fish (boiled).
Wholemeal and brown bread.	Chicken.
White bread should be toasted.	Meat should be minced.
Raw fruit.	Meat soups and broths.
Root vegetables (carrots turnips, parsnips, swedes *et cetera*).	Crisp toast and rusks.
	Crisply done bacon. Otherwise no fried food.
Peas and beans.	Milk, eggs, butter.
Sweets and chocolates.	Honey, seedless jam and jellies.
Jam with seeds or pips.	Fruit stewed, and fruit juices.
	Green vegetables puréed (cauliflower, cabbage, spinach).
	Potato (see note below).

Note.—Potato should be well boiled, not mashed, and limited to two tablespoonfuls.

Stewed fruit: A little daily, provided that no skins or pips are given.

Farinaceous puddings should be withheld for two or three weeks; as improvement begins, a gradual return should be made to cornflour, custard, ground rice, rice, and semolina.

cubic centimetres (two fluid drachms). The dose is two teaspoonfuls, to be taken five minutes before meals. The alkali promotes secretion of gastric juice and may help to loosen any mucus from the gastric mucosa; the rhubarb has a mild laxative action and at the same time improves the tone of the bowel wall; the *nux vomica* is given for its general tonic effect, while the gentian stimulates appetite. Mercury is also sometimes useful, and is best given as calomel, using small doses such as 0·015 gramme (one-sixth of a grain) thrice daily.

After-Treatment.

Generally an improvement begins to appear after three or four weeks of the above *régime*. At this stage a period of convalescence at the seaside for six or eight weeks is often invaluable; there is no better way of accelerating the improvement. While the child is away the routine of daily management and the dietetic precautions should not be relaxed; and generally, if drugs are being used, they should continue to be taken. Also at this stage, if anæmia has been a troublesome symptom, small doses of iron should be given; a useful form is the syrup of the iodide of iron, of which half a teaspoonful may be taken with the same amount of malt extract after meals. Iron should not be given during the earlier stage, as it is apt to be badly tolerated when the digestion is out of order. No attempt should be made to feed up the child, and the parents must particularly be warned against this. Cod liver oil, or one of the numerous preparations containing it, is a favourite prescription of the parents, who are anxious to get their child on, but it often affects the appetite adversely and is likely to aggravate the symptoms, so that it should not be recommended.

The various associated complaints, such as night terrors, nocturnal enuresis, or threadworm infection may also call for treatment, and a course of breathing exercises, or exercises to improve the tone of the postural muscles may be advisable in order to correct the child's stance. Equally, when these symptoms are complained of, the possibility that they may owe their origin to chronic digestive disorder should not be forgotten.

Reviews.

ELECTRO-SURGERY.

The use of high frequency currents now plays such an important part in many surgical fields that there exists a real need for a book such as "Electrosurgery", by Howard Kelly and Grant Ward.[1] In view of the importance of its achievements in opening up new surgical possibilities unthought of barely a decade ago, these authors have set out to deal with the entire subject with customary thoroughness from the historical, technical, experimental and therapeutic points of view.

The opening chapter on its early history is very interesting and full of detail, but the chronological order of the events leading to the evolution of the modern machine is rather confusing, and the lengthy description given of the work of Clark, of Philadelphia, on the subject is somewhat unnecessary. The terminology is a little difficult to understand. To avoid this, it might have been better to place the chapter on the physics of high frequency currents first. This chapter is an excellent one. The terms used in describing high frequency currents are clearly explained. The technical differences between high frequency currents given by the spark gap and vacuum tube machines, both "damped" and "undamped", and the principles of their construction in the various apparatus, are all fully set out, "not that it is essential for the surgeon to master all these electrical details at once, but to study them from time to time until his understanding of the principles is fairly complete, a matter of stick-at-it-iveness". Thus it is evident that diathermy, which literally means "heating through", requires a machine using high voltage, so that the low ampèrage will penetrate deeply into the tissues, whereas an adequate electro-surgical current or endothermy has an increased ampèrage and lower voltage, to deliver its destructive power on a tissue surface to a depth varying from a fraction of a millimetre in mild desiccation to several millimetres in heavy coagulation.

The biophysical effects of these various high frequency currents are next discussed, the classical work of W. T. Bovie being freely quoted. Electro-desiccation is dehydration produced by a uniterminal, "damped", high frequency current (Oudin); electro-coagulation reduces the tissues to a homogeneous mass by a biterminal high frequency current (d'Arsonval), which generates more heat and penetrates deeper; whilst acusection is true cutting with a "radio-knife" by a "radio tube" current of extremely high frequency and density. This current causes molecular disintegration of the tissues and seals off blood vessels and lymphatics and allows healing by first intention. All these effects are illustrated by microphotographs and supported by experimental studies, and a full description of an "ideal machine" for these three types of work is given.

The remaining chapters are given over to detailed description of the therapeutic application of electro-surgery in the various regions of the body. A word of advice is given against the careless use of its powerful effects "in the office" on out-patients; and operators are advised to adhere to the usual hospital and operating theatre routine, "whilst avoiding any extreme air of mystery, with the view of impressing the patient, a practice bordering

[1] "Electrosurgery", by H. A. Kelly, M.D., LL.D., F.A.C.S., and G. E. Ward, M.D., F.A.C.S.; 1932. Philadelphia: W. B. Saunders Company; Melbourne, Brisbane and Christchurch: James Little and Son. Super Royal 8vo., pp. 305, with 382 illustrations. Price: 45s. net.

closely on quackery". Local anæsthesia and pre-medication are advised where possible; and no dangers have been encountered from the administration of ether by the open method, provided the wet mask is removed from the operative field.

In the chapter on oto-laryngology, that much debated subject, "the electrosurgical removal of tonsils", receives excellent treatment and the various techniques are fully described. We congratulate the authors on their conclusions that: "*Imprimis*, electrosurgery by no means replaces the well established surgical enucleation, except in selected cases."

The chapter on the thorax, by Aycock, is a splendid exposition of the value and technique of thoracoscopy, but no mention has been made of the recent application of electro-surgery in the operative treatment of lung abscess and in the operation of lobectomy. In the abdomen it has been found capable of allowing bloodless incision of liver, spleen and kidney to be performed, and its use in the separation of adhesions should go a long way towards preventing their recurrence.

The chapter on urology contains all the latest work and references on the "transurethral electrosurgical resection of the bladder neck and benign hypertrophy of the prostate", together with a description of the various "resectoscopes" and their technique; but the special section on bladder tumours by Hugh Young is rather unconvincing from the electro-surgical point of view. Whilst agreeing with the value of electro-surgery in the treatment of *pruritus ani* and hæmorrhoids, we do not feel disposed to concur with the authors' claim that: "Radium is, *par excellence*, the sheet anchor in the treatment of rectal adenocarcinoma", even with a colostomy and aided by preliminary electro-surgery. Oh shades of Miles and Mummery, with their statistics for abdomino-perineal and perineal resections! The section on the central nervous system is a good one and ably demonstrates how electro-surgery, in the hands first of Cushing, the pioneer, and later of many others, has made it "possible to extirpate tumours so inaccessible that their attempted removal would have been regarded as foolhardy in the extreme".

The other chapters deal with the electro-surgical treatment of boils; carbuncles and various skin tumours, especially hæmangiomata; cancer of the oral cavity, especially after the use of radium; the thyreoid gland; breast, various gynæcological conditions; and its association with irradiation in *lupus vulgaris* and cancer of the face.

A complete bibliography is appended covering each chapter. The illustrations are numerous, very suitable and excellently reproduced. The subject matter is well set out and printed in readable fashion, despite a few such anomalistic spellings as curet and maneuver.

EMBRYONIC TISSUE AND REJUVENATION.

METHODS adopted in the search for the long-sought elixir of youth vary according to what we may perhaps be bold enough to call fashion, even in the world of medicine. Those based upon the furnishing of an additional supply of sex hormone are well known. More recent are the trials of other means, and much information concerning the alleged hormonal substances contained in embryonic tissues is given in the book published by N. E. Ischlondsky this year, entitled "Sécrétion Interne et Régénérescence".[1] Dr. Ischlondsky calls the extract he has prepared an internal secretion, though this is a term of doubtful propriety. He extracts the embryonic spleen, brain, liver, gonads and bone marrow and obtains a product which he administers by injection. The process of extraction is described only in moderate detail, and though the author states that there is every need for great attention to detail, his description is more general than technical.

He has studied the effects of this extract on old and ailing animals, as well as the human subject of senile, degenerative or morbid processes. In senile animals he claims to produce a well marked regression of the sign of decrescence due to age, as judged by such evidence as posture, weight, reaction to irritation, longevity, and capacity to acquire conditioned reflexes. He further describes improvement in tuberculous animals and naïvely tells how this part of his research was due to the dishonesty of his animal vendor who sold him animals previously infected in other laboratories in lieu of healthy specimens. The section dealing with the human clinical observations is by far the longest in the book, but unfortunately for the author's thesis it is by no means convincing. He lays down an ambitious programme, essaying to evaluate the mental improvement by a study of the power to concentrate, to remember, to sleep, to write, and to fulfil certain requirements with regard to conditional reflexes which are not described in this book, but apparently in other work by the same writer. More purely physical criteria are furnished by such evidence as that afforded by blood pressure readings, blood counts, various chemical tests, and radiograms of skull and chest. The reader is, however, disappointed to find that the information given is often lacking in important detail, and even the protocols printed do not convince the critical judge. The case histories are meagre, and frequently no evidence is brought forward to exclude other therapeutic and psychic factors that might in themselves ameliorate the condition of the subjects of the treatment. The radiograms are not well reproduced, and are not at all striking proofs of what they are set up to prove. For example, it might well happen that an elderly person might have a dilated heart, with some degree of congestive failure, and that by rest and other treatment radiographic evidence of improvement could be truly presented without such adventitious aids as the exhibition of an embryonic extract. Cases are also described in which well marked clinical improvement was noted in patients suffering from asthenia resulting from various morbid changes.

In general the author has not sustained his case. Controls are admittedly most difficult to furnish in the study of any therapeutic agent, but the more convincing the controls, the more convincing the research. Dr. Ischlondsky is an optimist. He claims that his work clarifies such problems in medicine as the connexion between the sclerosing changes in the hypophysis with certain forms of epilepsy, the biological significance of the process of calcification in tuberculosis and arteriosclerosis, and the relations between the endocrine formula of the organism and general immunity. In the light of recent advances in our knowledge of the endocrines and of hormones nobody should reject any possibility: perhaps there are possibilities in embryonic tissue, but they need an advocacy and a proof alike unexceptionable. However, those interested in this question will find a mass of information in this book, and from it they may draw their own conclusions.

[1] "Sécrétion Interne et Régénérescence", by N. E. Ischlondsky; 1933. Paris: G. Doin and Company. Royal 8vo., pp. 342, with illustrations. Price: 90 fr.

VITAMINS AND FOOD.

MEDICAL practitioners may be excused for thinking that the book "Food, Health, Vitamins" is for them too elementary in its aim, considering that it is written for the general public,[1] but even those practitioners who keep abreast of the literature may fail to see the wood for the trees.

It is said that only a man who has completely mastered his subject is able to write a sound summary. If this is so, this small book of little more than a hundred pages may be recommended with confidence, as the authors are R. H. A. and V. G. Plimmer. The main theme is, of course, vitamin deficiency, a subject in which such rapid progress has been made in recent years that even active

[1] "Food, Health, Vitamins", by R. H. A. Plimmer, D.Sc., and V. G. Plimmer; Fifth Edition; 1932. London: Longmans, Green and Company. Crown 8vo., pp. 155. Price: 5s. 3d. net.

research workers, such as are the authors, might be excused for putting too great emphasis on detail and for failing to keep a firm grasp on the basic principles. This, however, they have not done; on the contrary, they have correlated recent progress, and as a result have given a new orientation to the subject.

Knowledge has gradually accumulated which has led to a recognition of the importance of shortage of vitamins as distinct from almost total absence of these entities, which was previously the principal object of study. As a result of this change of attitude, the authors stress the point, first discovered by experiments on animals and later confirmed by clinical tests, that shortage of vitamin B_1 is practically universal in civilized communities, not excluding wealthy members thereof, and that, in addition, the vast majority of those who are not rich suffer from pronounced deficiency of vitamins *A*, *C* and *D*.

The quantity of vitamin B_1 present in such foods as are usually eaten is insufficient to compensate for its absence in white flour, polished rice, pearl barley, sugar, jams *et cetera*, and consequently it is urged that, until such time as the removal of the germ of cereals is abolished by enlightened public opinion, everyone, without exception, should include in their dietary concentrated extracts of the vitamin which has been separated during the process of machine-milling, or otherwise make certain that their total calories are adequately balanced by a sufficient quantity of those foods which have been found to be extra rich in vitamin B_1.

Loss of appetite, indigestion, constipation, bouts of diarrhœa alternating with constipation, loss of weight, retarded growth in childhood, weakness, loss of vigour, headache, anæmia, slight œdema, unhealthy skin, subnormal temperature, susceptibility to infection, and various heart symptoms are all stated to occur as a result of shortage of vitamin B_1, and it is suggested that the chances of acquiring visceroptosis, *arthritis deformans*, appendicitis, and even cancer are increased as a result of the presence of this deficiency disease in a minor degree, that is, chronic vitamin B_1 deficiency.

This list may read like that of an advertising quack, but the fact remains that Lister's work produced a revolution in surgery no less surprising than would result in medicine if the statements made in this book should prove to be correct.

The authors have not limited their discussion to vitamin B_1, but have described the effect of deficiency of all the other vitamins also. It has been suggested that slight shortage of vitamin *C* over a long period may be responsible for *pyorrhœa alveolaris*.

Notes on Books, Current Journals and New Appliances.

ROYAL PRINCE ALFRED HOSPITAL PERINEORRHAPHY TABLE.

By H. H. Schlink, M.B., Ch.M. (Sydney), F.R.C.S.,
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Most surgeons have experienced difficulty with the lay-out of their instruments when performing operations on patients in the lithotomy position, especially those on the perineum, cervix and rectum. Many side tables have been designed to overcome this difficulty, but most are unsatisfactory, and a good many surgeons have discarded tables and use a tray which they place on their raised knees during such operations. The position, however, is awkward and does not allow of free movements. To obviate the drawback, I designed a table which, for experimental purposes, I first had made of wood fifteen years ago.

Figure I shows the table with the subsequent modifications which I made. It consists of a nickel-plated table top with a small and a large concave gap, the whole surrounded by a raised ledge to prevent the instruments being brushed off. Into the smaller gap a container for blood and used swabs fits by means of a slot under the table top and can easily be removed for sterilizing. Swab

Figure I.

and lotion bowls are fitted in swivel rings which turn in under the table top when not in use. The whole is on adjustable tubular steel legs mounted on rubber-tired, easy-running castors. There is a portable model in which the legs fold under the table. When the table is rolled into position the Auvard speculum fits into the container under the lesser concave gap. The larger gap is designed to allow the operator to sit close up to the perineum.

Figure II shows a convenient lay-out of instruments for a perineorrhaphy operation: Needle holder, Bonney's forceps, scissors and knife on the near right, artery forceps *et cetera* on the far right, Moynihan's and Duval's clips on the near left, and other instruments on the far left. Clean swabs are placed in the right and saline solution in

Figure II.

the left bowl. The great advantage is that all instruments are accessible and in front of the operator. By keeping to a constant lay-out the operator is able to pick up the instrument needed without removing the eye from the wound. The attendant, not having to pass instruments, can concentrate on threading needles, using the sucker and swabbing. Another great advantage is that the thread is not pulled out of the needle if it is necessary to place the needle-holder on the table when using a continuous suture.

The table is comparatively inexpensive and gives great comfort to the surgeon when performing operations on patients who are in the lithotomy position.

The Medical Journal of Australia

SATURDAY, OCTOBER 28, 1933.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

THE KING'S COLLEGE HOSPITAL NUMBER.

This issue of The Medical Journal of Australia contains nine articles specially contributed by members of the staff of King's College Hospital, London. It is several years since an issue was devoted to contributions from overseas; on that occasion practitioners from Canada sent articles as a result of representations made to them by Dr. N. D. Royle at the request of the Editor. On this occasion readers are indebted to Dr. R. Graham Brown, of Brisbane, who, during his visit to England to attend the Centenary Meeting of the British Medical Association, interested Mr. V. E. Negus in the work of this journal. Mr. Negus will probably be best known to medical practitioners as the author of that standard work, "The Mechanism of the Larynx". After some correspondence with the Editor, Mr. Negus kindly undertook to prevail upon members of the King's College Hospital staff to put pen to paper and to give Australian practitioners the benefit of their experience in different branches of medicine. The suggestion was made that the authors might bear in mind the needs of the general practitioner. Not only have the authors complied with this request, but they have covered a wide field. Every practitioner in the Commonwealth will find something in these articles to help him in his understanding of special problems and in his daily work.

Mr. J. Myles Bickerton's article on squint will be read with interest by ophthalmic surgeons, and general practitioners should be impressed with the need for early treatment and also with the importance of fusion training—subjects that have been mentioned comparatively recently in these pages. Mr. E. B. Clayton's remarks on the after-treatment of fractures will be read with interest by all Australian practitioners who treat fractures. In Dr. East's paper special emphasis should be laid on what he has to say about the preliminary use of morphine and on the performance of venesection before the time of its usefulness has passed. Mr. John Everidge has rendered a service by his discussion of transurethral technique for bladder neck obstruction. Since McCarthy's work was published no reports have appeared in this journal from the pen of Australian urologists who have used McCarthy's method. It is important to note that Mr. Everidge does not think that transurethral methods are likely to displace the operation of suprapubic prostatectomy. Dr. Gilliatt's paper on delay in labour summarizes the position admirably and will be useful to all general practitioners. Mr. John B. Hunter makes quite clear both the differences between pneumococcal and streptococcal empyemata and the indications for treatment. Equally important is his statement that no case of chronic empyema should be allowed to drift on without an attempt being made to remedy the condition. Dr. R. D. Lawrence's paper on the care of insulin diabetics will be welcomed by general practitioners and physician-specialists alike. His writings are well known and he is one of those who enjoy authority on the subject of diabetes. Both Mr. Negus and Dr. Wilfrid Sheldon deal with subjects that confront all medical practitioners sooner or later.

These articles from King's College Hospital are more than contributions that will interest and be useful to our readers. They are an expression of goodwill from leading practitioners in Great Britain, goodwill to this journal and to the practitioners of Australia. They will surely cement the ties that bind Australian to English medicine. In

offering the sincere thanks of the journal and its readers to Mr. Negus and his colleagues for their united efforts, we would give expression to the hope that many of them will be among the delegation that will visit Australia when the British Medical Association holds its annual meeting in Melbourne in 1935.

Current Comment.

AMYLOIDOSIS.

Amyloid disease is known also as lardaceous or waxy disease. The term "amyloid" is not apt, as the substance contains nitrogen and is more allied chemically to albumin or gelatin than to starch. It has been termed lardacein—it is not a simple degeneration product and affected organs are enlarged. It follows prolonged suppuration in conditions of chronic sepsis, syphilis, tuberculosis of bones, joints and lungs and empyema; but it also occurs in syphilis without suppuration and in the cachexia attending some forms of malignant disease. How these conditions act is not known. It is seen most often in the spleen, kidneys, liver, intestines, stomach and panacreas and, with diminishing frequency, in the suprarenals, lymph glands, thyreoid gland, aorta, ovaries and uterus. Amyloid tumours of the larynx are rare. They may be a local manifestation of amyloidosis or may replace pre-existing tumour cells. Such tumours may be mistaken for malignant growths or syphilitic gummata. Amyloid disease may be seen in the conjunctiva, generally, but not always, following trachoma. Adamük regards the material as a deposit between the connective tissue cells and fibres and not a product of cell degeneration. In general the tissues invaded are firstly the walls of the blood vessels, especially the inner and middle coats with the muscular fibres; secondly, connective tissues and lastly, if at all, the gland cells of an organ. The organs affected are of firm consistence. The amyloid substance is firm, colourless and translucent, resisting putrefaction and chemical action. It is insoluble in dilute acids and very slightly soluble in gastric juice at body temperature. With some colouring agents it gives special reactions. With iodine a rich brownish-red or claret colour is formed, changed to dark purple on the addition of dilute sulphuric acid. Methyl violet gives a red or bluish-pink tint, the surrounding healthy tissue being stained blue. When the spleen is affected it is large and hard, with a typical "sago spleen" appearance. An affected liver becomes greatly enlarged, but painless and not tender. There is no jaundice and if ascites and anasarca exist, they are attributed to coexisting kidney disease. A slight amount of amyloid disease may be seen in chronic nephritis apart from profuse suppuration or cachexia.

E. G. Bannick, J. M. Berkman and D. G. Beaver have described three unusual cases of diffuse amyloidosis.[1] The first case was one of lymphosarcoma of Hodgkin's type with healed duodenal ulcer and pseudomembranous entero-colitis. The spleen, kidneys, liver, suprarenals, ileum and colon were the sites of amyloidosis. The lymphosarcoma was probably the basis of the amyloidosis. The diagnosis of the amyloidosis was correctly made after various forms of nephritis and nephrosis were excluded. Bence-Jones proteinuria was also excluded, but the result of the congo red test for amyloid disease was positive and hepatic function tests indicated extensive liver implication. In the second case autopsy revealed acute tuberculous degeneration of the kidneys and healed tuberculosis of the hilar glands of the lungs with atherosclerosis and cholelithiasis. There was amyloidosis affecting the spleen, liver, suprarenal glands, myocardium, lymph glands, kidneys, pancreas, thyreoid gland, thymus, lungs, stomach and other organs. There was hypertrophy of the liver and spleen with ascites, hydrothorax and œdema of the lungs. What determined the amyloidosis was not certainly ascertained. No congo red test was employed during life; at the autopsy it gave its characteristic reaction. The methyl violet test was only partly successful; it did not disclose the usual degree of metachromatic reaction given by amyloid substance. Iodine alone or followed by sulphuric acid did not give a typical reaction. In sections it failed entirely and in the fresh state the gross iodine reaction gave a black or dark brown colour, but not the typical reddish brown. The diagnosis of amyloidosis was not made. There was no evidence of suppuration. The terminal illness was characterized by renal, suprarenal and hepatic insufficiency. The diffuse hyaline amyloid-like substance developed without apparent cause. Other hyaline substances have a marked affinity for congo red, and in this case there was no chemically perfect amyloid substance but some allied or intermediate body. The third case revealed carcinoma of the stomach, evidently of long standing. It was of a medullary type of carcinoma simplex, with peculiar acidophilic staining of the cytoplasm. It probably was concerned with the pathogenesis of the amyloidosis. There was also hypoplasia of the heart with calcareous deposits on the mitral valve, hydrothorax and slight œdema of the lower extremities. Amyloidosis involved the liver, spleen, kidneys and suprarenals. There was no suppuration. Diffuse amyloidosis and amyloid nephrosis were tentatively diagnosed. Lipoid nephrosis, the nephrotic stage of chronic glomerulo-nephritis and cardiac dropsy were excluded. No congo red test could be carried out during life. At the autopsy typical staining reactions were elicited.

Bannick and his colleagues state that diffuse amyloidosis can usually be diagnosed, if suspected. The congo red test is valuable in the diagnosis. It had previously been noted by H. Bennhold that patients with extensive amyloidosis, particularly of the liver, almost all gave positive tests with congo

[1] *Archives of Internal Medicine*, June, 1933.

red; but no reaction may be obtained when the deposits are not extensive. Bannick insists that amyloidosis should be considered in all unusual cases of hepatic, renal, suprarenal or splenic disease, even in the absence of any obvious cause for amyloidosis. In each of the three cases reported by Bannick suprarenal inefficiency was noted clinically and the autopsies revealed extensive amyloidosis of those structures. Hypotension was marked in all and in two cases an Addisonian crisis was simulated. Abnormal pigmentation was not seen.

The explanation of amyloidosis is still unknown. Many years ago Dickinson suggested that it resulted from the loss of alkali to the body by the draining away of abundant pus. This view is not acceptable, especially considering the cases complicating malignant disease and syphilis, in the absence of suppuration, and some cases of limited amyloidosis of the kidney with chronic nephritis. Another matter requiring elucidation is the infrequency of amyloid disease in Australia in spite of the occurrence of profusely suppurating diseases.

THE OPERABILITY OF CANCER OF THE LUNG.

During recent years great interest has been taken in primary carcinoma of the lung. Not only have the methods of diagnosis vastly improved, making the recognition of this form of new-growth possible before too serious an extension has occurred, but the condition itself appears to be increasing in absolute frequency. It would, however, be a mournful academic task to describe and define the early signs of a malady entirely beyond hope of relief, and the importance of early correct diagnosis centres round the possibility of successful treatment. Even a few years ago to mention the operation of lobectomy or partial resection of a lung was almost to invite remarks about an appalling mortality. But now the position is very different. Thoracic surgeons can explore the thorax in safety and perform radical operations with what is, considering the extensive and serious nature of the procedure, a relatively low mortality risk. Therefore the radical removal of part of a lung for cancer is occasionally a surgical possibility. We must not overlook the possibilities of radiation therapy also, the greatest promise of which seems to be in conjunction with some form of surgical approach.

H. A. Carlson and H. C. Ballon contribute a recent article on the possibilities of surgery in lung carcinoma.[1] Their communication is largely based upon autopsy studies, a method of approach which is to be commended as giving positive and, within its own ambit, accurate information. They studied twenty-six cases in order to arrive at conclusions concerning the best diagnostic methods, to correlate the autopsy findings as regards metastases with the clinical signs, and to estimate the possibilities of surgical treatment.

[1] *The Journal of Thoracic Surgery*, April, 1933.

The histories of the patients revealed that the commonest symptoms were cough, pain or discomfort in the chest, dyspnœa, the presence of sputum and general asthenia as manifested by loss of weight, weakness and so forth. The continuous and disturbing nature of the thoracic pain or discomfort felt by these patients was very noticeable, and, taken in conjunction with cough, dyspnœa and hæmoptysis, helped to establish a tetrad of symptoms of distinct diagnostic value.

They also refer to the chronicity of symptoms, suggesting abscess, tuberculosis, unresolved pneumonia *et cetera;* this is well known. X ray examination suggested the correct diagnosis in not quite half the cases, and the use of a contrast medium of iodized oil was valuable in a number. With regard to the latter method, the authors issue a warning against a hasty diagnosis of blocked bronchus, for what appears to be a block may be merely defective filling. Bronchoscopy they aptly describe as the only method that permits the making of an early positive diagnosis. Clinicians who are still inclined to look askance at this method, which alone enables a skilled observer to visualize the bronchial tree, should take note of this. Other special methods on the value of which they specially remark are the diagnostic induction of pneumothorax and exploratory thoracotomy. The difficulty in arriving at an accurate diagnosis is illustrated by the striking fact that of these twenty-six patients who actually came to autopsy only seventeen had their ailment correctly diagnosed. Some of these persons were in an advanced state of illness on entering hospital, and complete examination was practically out of the question. The investigations made into the occurrence of metastatic deposits showed that bronchial tumours grow slowly, and that, though metastases may arise early and even give rise to recognizable signs in distant organs, as a rule secondary spread is not common till the terminal stages are reached. This is well known, of course, for though signs of metastases should be looked for in the thorax, liver and central nervous system in particular, the disease may run its course and remain localized. Carlson and Ballon have collected figures from the literature indicating the results of surgery. There are very few successes recorded as yet, as might be expected. Bronchoscopic removal one would assume to be unlikely to be successful; indeed only one success is reported. Successful radical operations have been few, but they are encouraging. To know that even a few patients have survived for months or even for one or two years and that some remain well is cheering. Cautery pneumectomy seems to be the most promising radical procedure, but there also appears to be hope in the implantation of radon seeds, either through a bronchoscope, or more logically perhaps, after performance of thoracotomy.

The surgical position is certainly depressing on the whole, but there is a little light ahead; methods of treatment are improving; accurate diagnostic methods must be further improved and used.

Abstracts from Current Medical Literature.

PÆDIATRICS.

Non-Protein Nitrogen of the Blood in Health and Hepatic Disease.

M. T. TINDAL (*Archives of Disease in Childhood*, February, 1933) holds that the normal liver should prevent flooding of the blood with urea after a high protein meal, while the diseased liver should permit the non-protein nitrogen of the blood to rise to an abnormally high level and remain elevated for an abnormally long period. The author first gave glucose with water and then water alone to each of five children suffering from valvular heart disease, chorea and renal calculi. A fall occurred in the fasting level of the non-protein nitrogen in each instance. A similar fall occurred in continued fasting, when neither glucose nor water was given, so that the fall must be due to continued nitrogen starvation. In the author's investigation the finger blood was taken after twelve hours' fast, and the non-protein nitrogen estimated by Folin and Svedberg's micro method. The children were then given 15 grammes of urea in 120 cubic centimetres of water and further blood samples obtained after 30, 60 and 120 minutes. Twelve convalescent subjects were selected, in none of whom was there any reason to suspect a disorder of metabolism. They were aged from four to twelve years and none was receiving any drug treatment. The fasting non-protein nitrogen was found to lie between 18 and 40 milligrammes *per centum*, and in all cases a definite rise was observed within half an hour of the ingestion of the urea. The smallest increase was 4·6 milligrammes *per centum* and the largest 35 milligrammes. In nine instances the curves were continuing to rise even after the first hour. The other three curves were in the downward trend. By two hours all were falling, and only three were more than 20 milligrammes above the fasting level. It appeared that the size of the dose of urea in relationship to the age of the child was of no moment. The author describes the normal curve in children as one which reaches its maximum within two hours of urea ingestion. Thirteen children in whom liver disease was known or suspected were treated by the same procedure. The fasting level lay between 26·7 and 52·6 milligrammes. One hour after the urea the values remained at practically the same value as at thirty minutes. At two hours, six had risen more than 20 milligrammes. Three hours afterwards three were continuing to rise. By four hours all had begun to descend. The three characteristics of the pathological curve are: (i) the higher average non-protein nitrogen; (ii) the slow rise; (iii) the height and prolongation of the curve. To eliminate the possibility of a kidney factor, urea concentration tests were performed with satisfactory results. The author suggests that the high fasting level is due to an hepatic inability to deal with the products of endogenous protein breakdown, and that the delayed rise is due to some associated defect of absorption from the bowel, while the height and prolongation of the curves may be due to the diminished power of the diseased liver to retain and store the non-protein nitrogen.

Soya Bean Flour in Infant Feeding.

GENEVIEVE STEARNS (*American Journal of Diseases of Children*, July, 1933) has investigated the nitrogen, calcium and phosphorus balance of infants on soya bean flour. Soya bean flour is used as a milk substitute in milk-sensitive infants. Soya bean flour is rich in protein, but of a poorer quality than cow's milk. The beans are rich in potassium and magnesium, but contain very little sodium, calcium and chloride. Phosphorus is very much the same. It is customary to add sodium chloride and calcium to a soya bean feeding, while the caloric content is increased by 20% to 22% by the addition of oil and sugar. Cod liver oil and a source of vitamin *C* must also be given. The results of comparison of nitrogen, calcium and phosphorus retention between infants fed on milk and on soya bean were briefly as follows: An increase in the relative intake of nitrogen and calcium as compared with the phosphorus intake resulted in an insufficient retention of phosphorus. Under these conditions the urinary excretion of calcium was tremendously increased and the urinary phosphorus markedly decreased. Excessive excretion of calcium in the urine is accompanied by a shift in the mode of excretion of other bases, decreasing the excretion in urine and increasing the fæcal excretion. The altered calcium metabolism does not seem to affect the retention of fixed bases other than calcium. The substitution of di-calcium phosphate for calcium carbonate in the soya bean food improved the relative retentions of nitrogen, calcium and phosphorus. The author concludes from her observations that the modified soya bean food is a satisfactory food for infants.

Diphtheria of Glans Penis, Scalp and Fauces.

P. DAHR (*Münchener Medizinische Wochenschrift*, June 16, 1933) states that atypical localized diphtheria is difficult to recognize, since the only proof is bacteriological. Rarer than faucial, nasal or conjunctival diphtheria is true skin diphtheria and diphtheria of the middle ear. The early recognition of such true but atypical localized diphtheria is very important for the specific treatment of the sick and for the protection of their surroundings. The author records three cases in one family with true diphtheria; each one had a different localization. First of all a boy, aged five years, became ill with a balanitis diphtheria recognized to be virulent by bacteriological tests and inoculations. From his clinically healthy pharynx, virulent Klebs-Löffler bacilli were later obtained. Shortly afterwards his brother, aged one year, developed diphtheria of the skin of the scalp with similar positive bacteriological findings. A little later the sister developed a true faucial diphtheria. The whole four diphtheria groups from these three children showed similar characteristics culturally, microscopically and also in their virulence.

The Hæmolytic Streptococcus as the Cause of Acute Rheumatism.

H. J. GIBSON, W. A. R. THOMPSON AND D. STEWART (*Archives of Pediatrics*, February, 1933) record work on a series of patients suffering from acute rheumatism along lines similar to those employed by Collis and Sheldon in London, together with an equal number of non-rheumatic controls. The patients observed were of all clinical types and were tested as regards their skin sensitivity to all types of streptococcus, including the hæmolytic variety and Dick toxin. The work of the authors does not add much support to the allergic theory of acute rheumatism. They emphasize that no claim has ever been made for the hæmolytic streptococcal skin reaction as specific in acute rheumatism, but that it is at best an index of previous infection with hæmolytic streptococci, whether associated with scarlet fever or non-rheumatic hæmolytic streptococcal infection, which tends to be negatived by the development of Todd's streptolysins in the blood and the natural tendency towards the establishment of Dick negativity in the skin. The incidence of positive reactors among controls was much higher than in Collis's series. This was possibly due to the fact that many patients and controls were taken from wards housing convalescents from ear, nose and throat operations. The authors' cases of rheumatism revealed rather more evidence of previous infection with the hæmolytic streptococcus than did the non-rheumatic controls. The paramount question of what peculiarity exists in the host or micro-organism to determine acute rheumatism and its particular form remains unanswered. Hæmolytic streptococci were found in twice as many rheumatic patients as controls. The authors state that this may mean nothing more than that the throat is a more favourable nidus for the persistence of the microorganism. They intend to pursue serological studies to attempt to decide whether these are chance infections in rheumatic patients subsequent to the onset of disease, or entry into hospital, or whether they are really ætiologically related to the rheumatism. Two further main features were the large percentage of positive skin reactions to hæmolytic streptococcal extract

among cases of chorea (90%). Reactions were much less to extracts of *Streptococcus viridans* and the γ streptococcus. As an aid to diagnosis or prognosis the skin reactions were of little, if any, value. At a convalescent rheumatic home the highest percentages of skin reactions and positive throat swabbings were obtained, yet no relapses occurred. It suggests that skin sensitization is an index of the hæmolytic streptococcus factor alone, that is, it is not an indicator of the reactive state in the patient necessary to produce the "rheumatic" response to such infection. No evidence has been produced by these authors which is contrary to the theory of hæmolytic streptococci as the cause of acute rheumatism. The finding of 25% "negatives" in rheumatic patients is comparable to that found in scarlet fever, which is known to be produced by a form of this organism.

ORTHOPÆDIC SURGERY.

Generalized Osteitis Fibrosa with Hyperparathyreoidism.

R. C. Elmslie *et alii* (*The British Journal of Surgery*, January, 1933) describe case histories of three patients suffering from generalized *osteitis fibrosa* associated with tumours of the parathyreoid glands. The cysts in the bone were explored and submitted to histological examination. In no case was there any evidence of decalcification of the bones and the margins of the cysts. There was no evidence of fibrosis of the marrow, and no giant cell tumours were found. Sixteen months after removal of the parathyreoid tumour large cystic cavities still existed, but were surrounded by denser bone. The serum calcium before operation was abnormally high in two out of the three patients, and high in the third patient. The amount of calcium taken with the food had no effect on the level of serum calcium. The serum phosphorus was abnormally low in all three patients. A diet which was low in calcium provided a more severe test of the ability to store it, and so a high urinary calcium on such a diet was more conclusive evidence of calcium metabolism. After removal of the parathyreoid tumour, serum calcium fell in all three patients to a level considerably below the normal, generally within three days, and it was shown that this was due to an increased retention of calcium as evidenced by the disappearance of excessive excretion in the urine. The phosphorus metabolism was little altered by the removal of the tumours. The authors noted that in patients with generalized fibrocystic disease, even when the evidence was sufficiently clear to justify the assumption that a parathyreoid tumour was present, it was not always easily found at operation. The tumour of the parathyreoid in one instance measured 3·0 by 1·5 by 1·5 centimetres. It was yellowish red in colour, encapsulated, and showed fibrous trabeculæ arising from the capsule, with many cystic spaces between. The cytoplasm of the cells was slightly basophilic, but in no instance were the oxyphile cells of Welch to be found. The authors suggest the possibility that hyperparathyreoidism may prove to be the cause of generalized *osteitis fibrosa*, of which the outstanding features are pains and aches in the limbs, spontaneous fracture, cyst-like areas of rarefaction in the bones, general rarefaction of the bones elsewhere, muscular weakness and wasting. Renal calculi may be present in such cases. They cite the evidence of hyperparathyreoidism with constant abnormally high serum calcium and abnormally low serum inorganic phosphorus. The output of calcium exceeds the intake even on a generous diet, with an especial increase in the urinary excretion of calcium, and even then the faulty metabolism may not be detected unless low calcium intakes are utilized and the high urinary excretion is demonstrated. The most constant abnormality in the three cases was the increased urinary excretion of calcium. The authors describe generalized *osteitis fibrosa* as a condition which is twice as common in women as in men, the onset usually being in adult life or adolescence with bone pain. Bending of the bones may occur from fracture or collapse of a cystic area. Muscular weakness and hypertonicity are common. Renal calculi are common, and when the onset of the condition occurs in young subjects there may be delayed general and sexual developments. Focal *osteitis fibrosa* seems to occur equally in males and females, the onset usually being in childhood or adolescence. Bone pain is absent or slight, except when fracture occurs. Bending of bone occurs particularly in the upper ends of the femora and tibiæ, but muscular weakness, wasting, delayed sexual development and renal calculi do not occur.

H. Cohen and R. E. Kelly (*ibidem*) describe a case of generalized *osteitis fibrosa* associated with tumour of the parathyreoid gland. The patient was a single woman, aged forty-eight years, who had had renal calculi and was complaining of swelling of the lower jaw. Radiographs of the mandible demonstrated a loss of normal texture with scattered, irregular, translucent areas, some of which were clearly defined. Similar appearances were presented in radiographs of the skull, right innominate bone and upper portion of both tibiæ. Hypercalcæmia varying from 13·0 to 17·4 milligrammes per 100 cubic centimetres was constantly present before operation and disappeared almost immediately after removal of the parathyreoid gland, which measured 2·5 by 1·5 by 1·0 centimetres and weighed 2·5 grammes. Three days later the patient showed definite signs of impending tetany, which disappeared after intravenous injection of 10 cubic centimetres of a 10% solution of calcium gluconate and 20 units of parathormone. There was no recurrence of the tetany after four injections, and the calcium phosphorus metabolism remained normal after operation.

Arthritis Gonorrhoica Tarda.

F. Wirz (*Münchener Medizinische Wochenschrift*, March 3, 1933) writes that in those cases of joint disease characterized by a slow, creeping onset with widespread local symptoms and with none or general symptoms (fever, lassitude *et cetera*) one thinks of the following joint diseases: chronic rheumatism, tuberculous joint, syphilitic joint, and non-bacterial diseases (static, endocrine or metabolic). In the author's investigations during the last years he found that a special gonococcal ætiology causes this disease (*arthritis gonorrhoica tarda*), which is quite distinct from the ordinary gonococcal arthritis. The latter type resembles closely an acute polyarthritis with marked general and local symptoms, and is a complication of acute or subacute gonorrhœa occurring within the first to the fourth week of the infection. Occasionally it disappears for a shorter or longer period, later reappears as a wandering arthritis, and it is now spoken of as a chronic arthritis, which is incorrect, since healing and recrudescences occur. Totally distinct are the cases of late gonococcal arthritis. Six of these cases were observed in the last three years; four occurred in men, one in a woman, and one in a girl. The symptoms closely resemble the remittent stage of gonococcal arthritis rather than the so-called gonococcal rheumatism. In this type the tissues are only very slightly involved and no or a very slight effusion may occur. The temperature is normal or at the most subfebrile; the interference with movement does not extend to the loss of use. The X ray picture is not very characteristic and in most cases a slight broadening of the shadow of the joint capsule is present. If the bone is involved, a moderate, mostly spotted, calcium deficiency is present, resembling tuberculosis, but the frequent periosteal bony deposits in the limbs, especially in the foot, suggest a more productive process. The positive genital findings were evident in most cases on the first examination without the patients having such symptoms. In two cases local and general provocation were used before proof of the disease was obtained. The characteristic feature is the late onset after the gonococcal infection (on an average seven years) and also that the lower extremities are attacked. Following anti-gonococcal treatment a rapid and complete healing occurs. In all cases of migratory joint disease one must consider the possibility of a gonococcal *arthritis tarda*, prove the diagnosis and treat the cause, for a good result may follow treatment.

British Medical Association News.

NOMINATIONS AND ELECTIONS.

THE undermentioned has been nominated for election as a member of the New South Wales Branch of the British Medical Association:

Ternes, Alfred Christopher, M.B., B.S., 1927 (Univ. Sydney), Portland.

The undermentioned have been elected members of the New South Wales Branch of the British Medical Association:

Anderson, John Ivor Harold, M.B., B.S., 1931 (Univ. Sydney), Coast Hospital, Little Bay.

Douglas, John Richard Steele, M.B., B.S., 1931 (Univ. Sydney), Newcastle Hospital, Newcastle.

Phillips, Gilbert Edward, M.B., B.S., 1929 (Univ. Sydney), Department of Surgery, University of Sydney, Camperdown.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

Office-Bearers of Sections.

THE following is the list of those who have accepted the positions of office-bearers of the several sections of the fourth session of the Australasian Medical Congress (British Medical Association), to be held in Hobart in January next. The list is not yet complete.

I. *Section of Anæsthetics.*

President: Dr. L. Lillies, Victoria.

Vice-Presidents: Dr. Hugh Hunter (New South Wales), Dr. Ellis Murphy (Queensland), Dr. Mathew Kasner Moss (Western Australia).

Secretaries: Dr. Cedric Duncombe (185, Macquarie Street, Hobart, Tasmania), Dr. G. A. Kaye (14, Collins Street, Melbourne, C.1).

II. *Section of Medicine.*

President: Dr. C. Bickerton Blackburn, O.B.E. (New South Wales).

Vice-Presidents: Professor D. W. Carmalt Jones (New Zealand), Dr. Eustace Russell (Queensland), Dr. A. W. Cowan (South Australia), Dr. J. A. Newell (Tasmania), Dr. L. S. Latham (Victoria), Dr. D. McWhae (Western Australia).

Secretaries: Dr. R. Wishaw (159, Macquarie Street, Hobart, Tasmania), Dr. C. W. Clemons (Cameron Street, Launceston, Tasmania).

III. *Section of Naval, Military, Air Force Medicine and Surgery.*

President: Dr. F. A. Maguire, D.S.O. (New South Wales).

Vice-Presidents: Dr. Gavin H. Cameron (Queensland), Dr. W. W. Giblin (Tasmania), Colonel A. T. White (Western Australia).

Secretary: Dr. R. M. W. Webster (Campbell Town, Tasmania).

IV. *Section of Obstetrics and Gynæcology.*

President: Dr. Hugh McLelland (Queensland).

Vice-Presidents: Dr. F. Brown Craig (New South Wales), Professor J. B. Dawson (New Zealand), Dr. R. T. Magarey (South Australia), Dr. R. G. Scott (Tasmania), Dr. B. K. Sutherland (Victoria), Dr. Dixie P. Clement (Western Australia).

Secretary: Dr. E. Brettingham Moore (149, Macquarie Street, Hobart, Tasmania).

V. *Section of Ophthalmology.*

President: Dr. L. Mitchell (Victoria).

Vice-Presidents: Dr. G. Antill Pockley (New South Wales), Dr. E. O. Marks (Queensland), Dr. A. L. Tostevin (South Australia), Dr. G. H. Hogg (Tasmania), Dr. J. Thompson (Western Australia).

Secretary: Dr. Bruce Hamilton (174, Macquarie Street, Hobart, Tasmania).

VI. *Section of Oto-Rhino-Laryngology.*

President: Dr. H. M. Jay (South Australia).

Vice-Presidents: Dr. Garnet Halloran (New South Wales), Dr. T. H. Price (Queensland), Dr. Stoddart Barr (Tasmania), Dr. G. A. D. McArthur (Victoria), Dr. H. B. Gill (Western Australia).

Secretary: Dr. B. Hiller (174, Macquarie Street, Hobart, Tasmania).

VII. *Section of Pædiatrics.*

President: Dr. S. F. McDonald (Queensland).

Vice-Presidents: Dr. Wilfred Vickers, D.S.O. (New South Wales), Dr. R. L. Thorold Grant (South Australia), Dr. G. E. Clemons (Tasmania), Dr. F. Kingsley Norris (Victoria), Dr. R. H. Crisp (Western Australia).

Secretary: Dr. A. W. Shugg (174, Macquarie Street, Hobart, Tasmania).

VIII. *Section of Pathology, Bacteriology and Cancer Research.*

President: Dr. J. V. J. Duhig (Queensland).

Vice-Presidents: Dr. E. F. D'Ath (New Zealand), Dr. D. L. Barlow (South Australia), Dr. John Ramsay (Tasmania), Professor H. A. Woodruff (Victoria).

Secretaries: Dr. J. H. B. Walch (171, Macquarie Street, Hobart, Tasmania), Dr. J. L. Groves (Launceston Public Hospital, Tasmania).

IX. *Section of Public Health, Preventive Medicine and Tropical Hygiene.*

President: Dr. J. Dale (Victoria).

Vice-Presidents: Dr. E. S. Morris (New South Wales), Dr. R. W. Cilento (Queensland), Dr. Percival T. S. Cherry (South Australia), Dr. G. Wade (Tasmania), Dr. R. C. E. Atkinson (Western Australia).

Secretary: Dr. C. N. Atkins (Macquarie Street, Hobart, Tasmania).

X. *Section of Surgery.*

President: Dr. Bronte Smeaton (South Australia).

Vice-Presidents: Sir John L. McKelvey (New South Wales), Dr. E. D. Ahern (Queensland), Dr. H. W. Sweetnam (Tasmania), Dr. B. T. Zwar (Victoria), Dr. L. T. Gillespie (Western Australia).

Secretaries: Dr. T. Giblin (Macquarie Street, Hobart, Tasmania), Dr. C. Craig (Brisbane Street, Launceston, Tasmania).

XI. *Section of X Ray and Electrical Therapy.*

President: Dr. H. R. Sear (New South Wales).

VicePresidents: Dr. V. McDowall (Queensland), Dr. J. S. Verco (South Australia), Dr. F. Short (Tasmania), Dr. K. S. Cross (Victoria), Dr. D. I. Smith (Western Australia).

Secretary: Dr. W. P. Holman (Saint Margaret's Hospital, Launceston, Tasmania).

Provisional Programme of Congress.

The following provisional programme of Congress has been drafted:

Monday, January 15, 1934.

9 a.m. to 4 p.m.—Registration of members at Tasmanian University Buildings.

3.30 p.m.—Garden Party, Government House, given by Sir Ernest Clarke.

8.30 p.m.—Inaugural meeting of Congress at Town Hall, Hobart. Address by Dr. D. H. Lines, President of Congress.
10 p.m.—President's reception at the Tasmanian Museum.

Tuesday, January 16, 1934.

9.30 a.m. to 12.30 p.m.—All Sections. Discussion on cancer and cancer research. Dr. D. H. E. Lines, President, in the chair, at the Main Hall, Philip Smith Teachers' College, University.
All day golf match (ladies), Kingston.
12.30 p.m.—Official photographs at University and Teachers' College.
2 to 4 p.m.—Sectional meetings at University.
8.30 to 9 p.m.—Mayoral Reception at City Hall, to be followed by Congress dance given by the Tasmanian members of the British Medical Association.

Wednesday, January 17, 1934.

9.30 a.m.—Sectional meetings.
2.30 p.m.—Sectional meetings.
Sectional dinners to be arranged by sectional secretaries.
Women graduates' dinner.

Thursday, January 18, 1934.

9.30 a.m.—All sections. "Hospital Problems in Australia." Dr. D. H. E. Lines in the chair.
2.30 p.m.—Sectional meetings.
8.30 p.m.—Popular lectures at Town Hall, delivered by Professor W. A. Osborne.
Sectional dinners.

Friday, January 19, 1934.

9.30 a.m. to 12.30 p.m.—Sectional meetings.
2.30 p.m.—Sectional meetings.
All day golf match (men), Rosny.
Evening.—Red Cross charity ball.

Saturday, January 20, 1934.

Afternoon.—T.A.J.C. races, Elwick.
Evening.—Sectional dinners.
Private entertainments.

Sunday, January 21, 1934.

All day excursion to National Park, by invitation of the Government of Tasmania.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on October 9, 1933.

The degree of Bachelor of Surgery (B.S.) was conferred *in absentia* upon Ronald William Grant Kelly, M.B. (London).

The gift of the equivalent of £200 towards the purchase of an electrical multiple stethoscope for the new Medical School was received with grateful thanks from the Western Electric Company (Australasia), Limited, Sydney.

The Royal Consul-General for Italy advised the Senate that it was the intention of the Italian Government to continue to provide for another four years after this year the subsidy now being paid to the University in connexion with the teaching of the Italian language.

The following appointment was approved: Miss Ruby Payne-Scott, B.Sc., as Temporary Physicist in the Department of Cancer Research.

The following Examiners were appointed for the forthcoming final degree examination in medicine:

Medicine: Professor C. G. Lambie, Dr. S. A. Smith.
Clinical Medicine: Dr. C. B. Blackburn, Dr. H. J. Ritchie, Dr. M. Lidwill, Dr. A. W. Holmes à Court.
Surgery: Professor H. R. Dew, Dr. J. C. Storey.
Clinical Surgery: Sir John McKelvey, Dr. A. Aspinall, Dr. H. R. G. Poate, Dr. H. S. Stacy.
Clinical Surgery (case taking only): Professor H. R. Dew, Dr. V. M. Coppleson.
Operative Surgery: Professor H. R. Dew, Dr. B. T. Edye.
Obstetrics: Professor J. C. Windeyer, Dr. P. L. Hipsley.
Clinical Obstetrics: Dr. Constance D'Arcy, Dr. A. J. Gibson, Dr. H. C. E. Donovan.
Gynæcology: Dr. R. L. Davies, Dr. C. Bowker.

It was decided to fix Thursday, October 26, as the date for holding the ceremony of commemoration of benefactors and combined religious service.

Correspondence.

PATHOLOGICAL EXAMINATION OF TUMOURS.

SIR: In your issue of June 17 attention was drawn to variations in the geographical dissemination of cancer of the breast and cancer of the tongue, and a note of warning sounded with regard to the limitations of unrestricted removal of tumour tissue for biopsy study in the former disease. Although Circular 716 of the Ministry of Health was quoted, Dr. L. B. Bull and Dr. B. S. Hanson, in their reply thereto, state: "As treatment becomes more efficient the objection to biopsy study, which appears to be mainly theoretical, will probably disappear."

The experience of a surgeon after a quarter of a century of active practice leads one to believe that the above objection, far from being theoretical, is of the utmost practical importance.

The spread of cancer cells being invisible to the naked eye, it naturally follows that clinical proof of the above contention must be restricted to those rare cases of lymphatic inflammation following almost immediately upon an exploratory incision.

Marmaduke Sheild, in his monograph "Diseases of the Breast", relates the case of a woman, aged fifty-four, who was admitted into Saint George's Hospital with a small lump in the axillary mammæ which had been incised. "A few days after the swelling in the axilla was thus opened, the skin round it became dusky red and hard like leather, and here and there 'warty lumps' appeared."

Similar cases are recorded in the literature, and two such cases have come under my notice, one of which followed exploratory incision.

The fact that recent incision for biopsy study is not usually followed by visible implantation or dissemination cannot be regarded as evidence that it does not occur. Further, in Dr. Bull's and Dr. Hanson's cases it would appear less likely to occur, as post-operative radiation is commenced "within an hour or so, or within twenty-four hours". The surgeon's problem, however, is much more complicated. Professor James Ewing (*Bulletin of the American Society for the Control of Cancer*, January, 1933) discusses the value of biopsy in the mammary cancer and arrives at practically the same conclusions as those enumerated in Circular 716, issued by the Ministry of Health. This paper is referred to in a short article, "Biopsy in Mammary Cancer", in *The British Medical Journal* of February 18, 1933.

Sampson Handley, in discussing the principles of the operative treatment of malignant disease in "Modern Operative Surgery", by H. W. Carson, writes: "Personally I believe that if a carcinoma is incised it is safer to ablate it at once rather than to wait for the wound to heal. The circulatory activity and inflammatory processes of healing must, I think, tend to favour rapid dissemination."

As the proof of the pudding is in the eating, let one ascertain what can be learned from post-operative results.

Table of Comparative Operative Statistics (1867 to 1913). Number of Cases "Cured".

Investigator, Name and Date.	Number of Cases.	Primary Mortality Percentage.	"Cured" Three-Year Limit Percentage.	"Cured" Five-Year Limit Percentage.
V. Winiwarter (Billroth), 1867-1875	—	23·100	4·7	—
Hildebrand (König), 1875-1885	—	7·200	22·5	—
Halsted, 1889-1894 ..	76	Nil	45·0	—
Schwarzkopf (Wölfler), 1895-1910	—	4·600	29·4	21·0
Halsted, 1907	232	2·500	38·3	—
Deaver, 1898-1913 ..	506	0·098	34·0	26·0

In an analysis of 338 cases operated on at Guy's Hospital and reported by C. Braine and G. Massie in *Guy's Hospital Reports* (1926), 54·1% of the patients were alive at the end of three years, and 28·4% at the end of five years.

Dr. Louisa Martindale (*The Lancet*, Volume I, 1931, page 229) has shown that adequate X ray treatment improves the three-year end results by 23% in early and 44% in later cases; the five-year end results by 23% in early and 40% in later cases; and the ten-year end results by 4% in early and 69% in later cases.

Dr. A. T. Nisbet, in reviewing the statistics of carcinoma of the breast taken from the records of Saint Vincent's Hospital, the Royal Prince Alfred Hospital, and his own private practice from 1928 onwards, stated at the Fourth Australian Cancer Conference (March, 1933) that the figures:

> show that our of three hundred and seventeen cases one hundred and ninety-nine (62·7%) have died in this short period.
>
> Of the one hundred and eighteen cases (37·2%) still alive, only forty-one (12·9%), or thereabouts, can really be said to have a favourable prognosis. The remaining seventy-seven have had some supraclavicular or other glandular enlargement, or have had secondary nodules in the skin about the scar or elsewhere, which have for the time being cleared up under deep X-ray therapy.

True, these results are reminiscent of the mid-Victorian era, and, rightly or wrongly, Dr. A. T. Nisbet castigates surgeons in no uncertain measure: "I am sorry to labour this point, but in my opinion surgery of cancer of the breast has failed, and failed very badly."

As the average duration of life in untreated breast cancer has been computed by Campiche and Lazarus Barlow to be about four years, it would appear rational to ask if these cases may not have been better off if treatment had not been instigated.

The modern surgical treatment of cancer of the breast is based on two factors: (*a*) the correctness of the operative method for its removal, and (*b*) the proper use of supplementary radiation.

Does the existing unsatisfactory state of affairs arise from: (*a*) A delayed recognition of the malady either by the patient or her medical adviser? (*b*) The employment of inadequate or unorthodox surgical methods, for example, delayed operation following incision for biopsy? (*c*) Inadequate or unsuitable supplementary radiation?

One would suggest that cancer be made a notifiable disease so that the Department of Public Health may introduce a follow-up system, study these problems, and direct treatment into those avenues most likely to prove increasingly successful.

Yours, etc.,

H. Rutherford Darling.

229, Macquarie Street,
Sydney,
October 10, 1933.

CHRONIC NEPHRITIS.

Sir: In an article on the treatment of chronic nephritis in your journal of the sixteenth ultimo Dr. Eustace Russell has cast doubt on the question of lead poisoning in childhood being a factor in the production of chronic nephritis in Queensland.

It is obvious that at the time of writing his article Dr. Russell had not made himself familiar with the recent work which has been carried out on the subject. Had he done so, I feel sure that he would not have dismissed the lead protagonists by merely commending them for their zeal, enthusiasm and pertinacity.

Although it is regretted that the Federal official inquiry into chronic nephritis in Queensland was terminated for reasons which are difficult to understand before any final conclusions were arrived at, the report contains a mass of evidence incriminating lead paint. Certainly the full report has not been published, but it can be perused by any interested persons at the office of the Commonwealth Health Department.

The following two extracts from the report are surely sufficient in themselves to make even the most biased opponent admit that there is at least a modicum of truth in the lead theory.

> 1. Of all etiological factors investigated, none was as commonly suggested as plumbism among 100 cases of the Brisbane series, 46 per cent. stating that they had been treated at an earlier period for "plumbism". In 30 of these cases hospital records indicated typical plumbism and in 16 indefinite signs.
>
> 2. Of 80 severe and typical cases of plumbism in childhood selected from the records of the Hospital for Sick Children, it was only possible to get in touch with 28. In 19 of these chronic nephritis was found at varying intervals after the recorded plumbism with paralysis. There was thus demonstrated here, as elsewhere, a definite relationship between the occurrence of marked plumbism and the subsequent occurrence of chronic nephritis.

It is my belief that the effects of lead on young tissue and its relations to arteriosclerosis and chronic nephritis have been so tragically demonstrated in Queensland that it should serve as a lesson to every country which has not prohibited the use of lead paints on verandas.

Fortunately, in their wisdom, the Queensland Government has asked for a report from an independent authority of high repute. When this is made available, it is hoped that the lead controversy will be terminated for all time.

Yours, etc.,

L. J. Jarvis Nye.

Wickham Terrace,
Brisbane,
October 10, 1933.

THE PROBLEM OF GASTRIC CARCINOMA.

Sir: Differences of opinion are inevitable; but there should not be much room for differences in regard to facts. It would seem that there is such a difference of opinion as to the usefulness of the benzidine test for occult blood in the fæces, when the diagnosis of gastric carcinoma, pernicious anæmia, and so on, is concerned. In these circumstances it may be useful for others and myself to retail our results in the use of this test. I do not do a great number of these tests (only about ten a year); but in my hands a positive result is unmistakable; even in the hands of the most careless and inattentive. As for a negative test, it also is unmistakable, though it might be possible for some people to torture themselves into believing that some cases are on the border-line. About five or six years ago, someone in one of the London hospitals did this test in 240 cases (all the cases in the hospital from whom a specimen of fæces could be obtained). None was positive, except cases of alimentary cancer. This was repeated, with the modification that no modification whatever was made in the patients' ordinary diet,

and the result was the same. These results were published. Ever since then I have never put a patient on a special diet before making the test, and in my hands the results are reliable. This effects a great saving of time and trouble. In my hands the results of the benzidine test (both positive and negative) are absolutely reliable.

Yours, etc.,

A. E. Taylor.

153, Fosters Street,
Dandenong,
Victoria,
October 10, 1933.

Sir: Since your anonymous correspondent "Pathologist" has now introduced Mallone's "Introductory Text Book of Logic" into the discussion, and as he states: "If less than two ounces of ox blood is taken, a positive Gregersen's test will not be given by the time the blood is passed in the fæces", I presume that he will in future refrain from advocating a preliminary "forty-eight hours of meat-free dieting, and, at least, twenty-four hours' diet free of green vegetables", owing to his belief: "the test in its essential is the detection of an iron-containing blood pigment"; otherwise he will be illogical.

But, according to my quasi-logical colleague, my "appeal to logic is unfortunate".

Yours, etc.,

H. Rutherford Darling.

229, Macquarie Street,
Sydney,
October 11, 1933.

THE USE OF IODINE IN THYREOTOXICOSIS.

Sir: Dr. Poate's interesting and stimulating article under the heading "The Use of Iodine in Thyreotoxicosis" really traverses a much wider field and raises many interesting questions. The fact that I have seen recently two instructive cases prompts this communication.

The first, a woman of thirty-nine, consulted a well known physician on account of tremor, nervousness, loss of weight and palpitation. Symptoms had existed for eight months. She was told that she must have immediate operation to prevent inevitable degeneration of her heart, that she was too ill to justify any delay. She was given Lugol's solution, minims five, to take three times daily, while she tried frantically, but vainly, to find the necessary surgical fee. Three weeks later she consulted me, declaring herself to be 100% better. Her basal metabolic rate was now +1. She returned to work and has remained well for six months.

The second, a woman of thirty-two, exhibiting marked nervous disturbances, sleeplessness and palpitation, after resting in bed and undergoing X ray treatment for two months, had her thyreoid removed. At the time of operation her basal metabolic rate was +10. She improved temporarily, but eight months later, after an attack of "influenza", all her symptoms reappeared and her condition was said to be as bad as ever. When seen ten weeks after the "influenza", there was no vestige of the thyreoid to be felt; the patient was in a highly nervous condition, with rapid pulse and many phobias. Thyreotoxicosis could not have played a large part in the production of her symptoms.

The surgeon, by nature a man of action, is apt to impress his view on the profession and public and so obscure the other side of the picture.

Animal experiment, which has taught us much, is prone sometimes to usurp a little arrogantly the domain of clinical observation.

For these reasons might I crave space to review the position briefly from the point of view of a perhaps over-conservative physician.

Clinical experience shows that every degree of primary hypersecretion exists, from slight, rapidly subsiding or becoming chronic, to fulminating, again rapidly subsiding, ending fatally or drifting into chronicity with or without exacerbations. Where previous enlargement of the gland exists, every phase from myxœdema at one end to acute hypersecretion at the other may be seen. Experience also shows that a very large number of patients recover under medical treatment, some even without it, and that the acuteness of onset is no criterion of the duration or severity of the attack. Such patients are not seen by the surgeon, whose views of the seriousness of the disease is correspondingly coloured. I well remember Sir Alexander MacCormick saying that he had never seen a case recover without operation.

Cases where no enlargement of the gland is found are uncommon.

Our knowledge of the causation of the hypersecretion is meagre. Hidden psychic trauma is much more common than is suspected; rarely, if ever, is sepsis answerable. To what extent the dyscrasia is primary and to what degree a response to outside stimulus is also uncertain, though evidence favours the more frequent occurrence of an external cause. Granted that hypersecretion is due to external stimulus, the gland will go on trying to do its duty however much is removed, and will endeavour to provide sufficient secretion for normal wants even after removal of the greater part of the gland during an exacerbation of hypersecretion. If it fails, myxœdema results, which, though airily dismissed by some surgeons, is an unsatisfactory condition. Nature can regulate the output of secretion to the patient's needs more accurately than can the physician the input of thyreoid tablets.

Inability to measure the severity and duration of the stimulus to over-secretion accounts for the cases that one sees temporarily "cured" by the physician, the surgeon and the radiographer in turn, and renders it impossible for the surgeon to promise cure by one operation unless he removes a very large portion of an essential gland.

Some surgeons are loath to admit that radiotherapy is of more than occasional value. My own experience is that in competent hands its value is so great that few cases need reach the surgeon, provided the patient can give time to treatment. Its drawback is the time involved. No improvement can be expected as the result of this form of therapy alone under three months. Its advantages are that the degree of secretion can be regulated more accurately, there is no risk to life and little of myxœdema. It is efficacious in cases that, on account of their gravity, have in the past been rejected by the surgeon.

Clinical experience also dispels the fear expressed by the surgeon of irrevocable harm done to the cardiac and other tissues by prolonged hyperthyreoidism. It is extraordinary how completely the majority of patients recover after operation or radiotherapy, even after very long periods of fibrillation.

The use of belladonna is hallowed by long usage. I have never seen it do harm and have frequently thought it did good. Arguments for and against any form of treatment based on an uncertain pathogenesis are often fallacious.

The recently rediscovered danger of iodides was a clinical fact known to our medical fathers. As students we were taught that patients with exophthalmic goitre were made worse by the continuous exhibition of iodides. This accounted for the scepticism with which some of us received the vaunted cure by iodine.

Many other interesting and debatable points emerge from Dr. Poate's paper, but cannot be dealt with in an already too long letter.

My object in writing is not in any way to belittle the enormous boon that surgery has brought to many sufferers from thyreotoxicosis; but is there not some risk that the pendulum may swing too far and that the mere ease and certainty of the operation may lead in some cases to the unnecessary removal of a necessary gland?

Medical treatment has a definite place and should always be given an adequate trial before resorting to surgical interference.

Yours, etc.,

Sinclair Gillies.

185, Macquarie Street,
Sydney,
October 12, 1933.

Obituary.

WILLIAM McELRATH MEEKE.

We regret to announce the death of Dr. William McElrath Meeke, which occurred on October 19, 1933, at Bondi, New South Wales.

WILLIAM ANGWIN EDWARDS.

We regret to announce the death of Dr. William Angwin Edwards, which occurred on October 22, 1933, at Sydney, New South Wales.

Corrigendum.

An error has occurred in the special article on the treatment of callosities, corns and warts, by Dr. Herman Lawrence, which appeared in the issue of October 14, 1933. At page 529, in the second paragraph of the first column, the sentence: "These needles are 0·2 millimetre (monel metal) in thickness and contain 0·5 milligramme of radium element" should read: "These needles are 0·2 millimetre (monel metal) in thickness and contain radium element equal to five milligrammes."

Diary for the Month.

Nov. 1.—Western Australian Branch, B.M.A.: Council.
Nov. 2.—South Australian Branch, B.M.A.: Council.
Nov. 3.—Queensland Branch, B.M.A.: Branch.
Nov. 6.—New South Wales Branch, B.M.A.: Organization and Science Committee.
Nov. 9.—Victorian Branch, B.M.A.: Special Council Meeting.
Nov. 10.—Queensland Branch, B.M.A.: Council.
Nov. 14.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
Nov. 15.—Victorian Branch, B.M.A.: Clinical Meeting.
Nov. 15.—Western Australian Branch, B.M.A.: Branch.
Nov. 21.—New South Wales Branch, B.M.A.: Ethics Committee.
Nov. 23.—New South Wales Branch, B.M.A.: Clinical Meeting.
Nov. 24.—Queensland Branch, B.M.A.: Council.
Nov. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
Nov. 29.—Victorian Branch, B.M.A.: Clinical Meeting.
Nov. 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. J. M. Sanderson has been appointed as Quarantine Officer at Esperance, Western Australia, under the *Quarantine Act*, 1908-1924.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xvi and xvii xviii.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officers.
DEPARTMENT OF PUBLIC HEALTH, SYDNEY, NEW SOUTH WALES: Medical Officers.
MUNICIPALITY OF HAMILTON, TASMANIA: Resident Medical Officer.
NEWCASTLE HOSPITAL, NEWCASTLE, NEW SOUTH WALES: Resident Pathologist.
PARRAMATTA DISTRICT HOSPITAL, PARRAMATTA, NEW SOUTH WALES: Junior Resident Medical Officer.
PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Medical Officers.
RENWICK HOSPITAL FOR INFANTS, SYDNEY, NEW SOUTH WALES: Honorary Officers.
ROYAL AUSTRALIAN NAVY: Medical Officer.
SYDNEY HOSPITAL, SYDNEY, NEW SOUTH WALES: Honorary Officers.
THE BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associated Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL are advised, in their own interests, to submit a copy of their agreement to the Council before signing. Lower Burdekin District Hospital, Ayr.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Combined Friendly Societies, Clarendon and Kangarilla districts. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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